A STUDY ON MADHUMEGAM

(DIABETES MELLITUS)

DISSERTATION

Submitted to

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BRANCH I – MARUTHUVAM

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CERTIFICATE

Certified that I have gone through the dissertation submitted by Dr.M.selvamani a student of final M.D(S), Branch-I Maruthuvam, Government Siddha Medical College, Chennai and the dissertation work has been carried out by individual only.

Place: Chennai
Date:

Professor & Head of the Department
Post Graduate Department
Branch-I Maruthuvam
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Chennai.

INTRODUCTION

The Siddha System of Medicine is one of the ancient system of Medicine and it was originated nearly twelve thousand years ago at Deccan, which is one of the most ancient geological formations in the world, since the down of history, the home of Dravids the oldest Indian ace. The Tamil which seems to be the oldest lanaguage of the so called Dravidian group of languages grew independently of the any language. The siddha System of Medicine is now mostly is in existence at Tamil Nadu, where the Tamil Language is the Mother Tongue.

The Siddha System of Medicine is having specific character and having specialty that it not only cures the disease, but also prevents the disease.

And also the Siddha System of Medicine not only cures the disease of the body, and also diseases of the mind.

“jâahj nehÍ‰ jâa¥ òçÍ«
kâkª Âutéœj kh®¡f¥ - Ãâia
crhé æa‰Wk¿ ÎŸstiu¡ f©lhš
krhÎ« ga¥gLk« kh”

“brŒÍ kâkª Âutêj e‹Kiwik
iaa äyhk y¿ªjnu - ita
äirkh ålbuåD« é©kUªj uh»
æirth® FuthŒ vd¡F

- njiua® bt©gh 227 & 228

ÚçUéid¡ Fz«

ÚçUéid¡ Fz¤ij Úa¿éç¤J¢ brhšnth«
`Úçid¥ bgU¡fbyh‹W ÚçidaU¡fbyh‹W
ÚçêÎlnd bfhšY« Ú®¡f£L éidfbs‹W
ÚâyKiu¡Fäªj ã®ãiw¡ Fz¤ij¡ nfshŒ

- njiua® kfh fçrš

Extensive details of the urinary diseases which manifest as excessive urination or decreased urination is explained by Therayar, one of the pioneers of siddha Medical system, as Neerinai Perukkal Noi and Neerinai Arukkal Noi. Madhumegam is classified under Neerinai perukal Noi.

MADHUMEGAM is a chronic metabolic disorder popularly called as "NEERIZHIVU" characterized by polyuria, which is sweet in taste and odour resulting in gradual diminution of seven udal thathukkal.

The word MADHUMEGAM in Siddha term closely resembles with "DIABETES MELLITUS" in modern Medicine.

The Greeks who know about it's prominent manifestation of persistent polyuria named the disease "DIABETES" means passing like a fountain or through siphon, "MELLITUS" means sweetness.

According to the nature of the urine voided by the patients the disease is classified into Twenty varieties in Siddha Texts. This classification is also based on the derangement of three humors i.e. vatham, pitham and kabam. Among them four varieties are due to vatham, six varieties are due to pitham and ten varieties are due to kabam.

This disease has been described by "Yugi Muni" in "Vaidhya Chintha – mani – 800".

The same disease is also stated in "Saraha Samhita" in Ayurvedham along with classification actiology and remedy for that. Siddhars described twenty types polyuria according to Mukkutram and described on the basis of colour of urine, consistency, smell, taste, weight, sedimentation etc.,

Madhumegam is one of the six varietities of polyuria. Which comes under Pitham derangement.

According to Yugimuni and Agasthiyar this disease is due to Karma that is hereditary, also due to dietic variations. According to “Nadinool” it is said to be to over indulgence of sex.

In modern clinical entitity “DIABETES MELLITUS” is closely resembles one of the variety of “Pitha Prametam” i.e. “MADHUMEGAM”. It is a metabolic disorder caused by absolute or relative defiency of insulin characterized by polyurine polyphagia, polydipsia, ketanuria, Hypokalaemia and cellular dehydration etc.,

AIM AND OBJECTIVES

AIM OF THE STUDY

The aim of the study is to evaluate the efficacy of the Siddha Medicine in the management of Madhumegam.

If the current trends continue the number of persons affected will be more than double from 140 million to 300 million in the next 25 years as per the WHO Report.

International Diabetic Federation estimates that atleast 1,77,00,000 people in the world suffer from Diabetes.

India currently considered as the diabetic capital of the world the number of people expected to have Diabetes by 2025 is fifty to seventy five million.

Fifty percent of diabetes are unware of their disease.

This clearly indicates that more persons turn to Siddha system seeking remedy. The management of diabetes mellitus has continued to the challenging especially among the recently diagnosed type II Diabetes. The traditional systems of medicines have in store, the treatment methods, drug and regimen for effectively combating Madhumegam.

These factors made the author to take up Madhumegam as the Dissertation work.

OBJECTIVE

1. To make a detailed study of various literature dealing with actiology, classification, clinical features, prognosis, complication, diet and treatment of Madhumegam.
2. To study Madhumegam in various literature in comparison with Modern science.
3. To carry out a clinical trial with two compound formulations one as chooranam and other a kudineer.
4. To understand the incident of the disease with reference to age, sex, thinaigal, paruvakaalam, socio economic conditions diet and family history.
5. To use siddha and modern parameters to confirm diagnosis, severity and progness of the disease.
6. The tests and investigation of Siddhar's diagnostic principle.
7. To evaluate the Bio – chemical and Micro – Biological features of the drug.
8. To undertake pharmacological and toxicological study of the drug.

REVIEW OF LITERATURE

SIDDHA ASPECTS – MADHUMEGAM

“j¡f jhuâ khå¤ njh®fŸ nfŸ
g¡f khry« g¤ÂU tifÍnk
e¡f ehaf‹ eha»¡ nfbrhš
ä¡f eªÂ és«Ã éÂ¤jnj”

\- njiua® thfl«

According to Therayar Vahadam the Universe consists of two essential entities, that is Matter and Energy which Siddhars referred to as shiva sakthi. Shive explained “Megarogam” to Sakthi. Here Nandhi explains its symptoms to the world for the benefit of the humankind. This clearly indicates that the existence of this disease is as old as human race.

Madumegam has its description in various literatures like Yugi Vaitheya Chindamani, Agasthiyar Gunavagadam.

Madhumegam comes under Neerinoi Perukkal Noi as mentioned in Therayar Maha Karisal.

“ÚçUéid¡ Fz¤ij Úa¿ éç¤J¢
brhšth«
Úçid¥ bgU¡fbyh‹W Úçid aU¡f
byh‹W”

\- njiua® kfh fçrš

Veru Peyar – Synonyms

Mega Neer, Vegu Moothiram, Ennippu Neer, Neerizhivu, Thittipu Neer, Pramegam

Mega Neer - Due to megarogam

Vegu Moothiram - Frequency and Urinary and large quantity of Urine passed.

Thithippu Neer / Ennipu Neer - The Urine is sweet in taste

- Noi Naadal

Neerizhuvu - Excess of urination

- Lifco dictionary

Pramegam - Sanskrit name for Madhumegam

- Sarabendra Vaithiya Muraigal

Iyal – Definition

Madhumegam is defined as "Large quantity and high frequency of urination, Derangement of the seven udal thathukkal and loss of weight". If the urine is heated honey odour is emanated from it.

- Noi Naadal

"Úçid¥ bgU¡f by‹W« Úçê éy¡fz‰nfŸ
ÚythçÂ ngh‰ F¡» Ú£L¡F Kiw jŸshF«
Úè TlhJ if, fhš Úykhéid neuhF«
Úšbrhdhîuå‹ _¢R Úrkh Ka§f§fh£L«"

- Therayar Maha Karisal

Abdomen distance like sea, Slurring of speech, peripheral neuritis, lassitude dyspnoea are the symptoms of Madhumegam.

As per Athma Rakshmirtham body becomes weak, weight loss, profuse, dryness of skin and tongue, excessive thirst, tiredness, excess sleep indicate the presence of Madhumegam.

- Athma Rakshamirtham

Noi Varum Vazhi :

Diet Habits:

"nfhija® fyé nghij
bfhG¤j Ûåiw¢Á nghij
ghJthŒ beŒÍ« ghY«
gçÎl D©Õ uh»š
nrhj gh© Lut ä¡f

R¡»y Ãunk fªjh‹
XJ Úçê nru
Î©bld t¿ªJ bfhŸns”
- mf¤Âa® 1200
“c‰gé¡F« ghš beŒah èiw¢Á fŸshš
cçirahŒ Û‹w‹dhš tUé U¤j
k‰gé¡F« gjhu¤j¤ jhš kJu t°jhš
kªj§fŸ jå‰ bghÁ¤jš ntfh¥ g©l«
F‰gé¡F§ Fëªj t‹d k§if nfhZo
F¿¤jã¤ Âiujé®j y¡»å kªj«”
- ô» it¤Âa Áªjhkâ

Excessive intake of food rich in carbohydrate and fat, red meat, sweet food, raw food and sleeplessness give raise to Madhumegam, quotes Agathiyar and Yogi Munivar.

Sexual Indulgence :

“f‹å ka¡f¤jhš f©oL nkfnk”
- eho üš ÂU_y®

“»uªÂ¥ ò©âuz nkf¡
Ñrf bd‹Dª J‹kh®¡f‹
mUªjÂ ba‹D« ghŠrhè a‹idia¡
f©Q‰whns”
- kU¤Jt ghuj«

According to Thirumoolar and Therayar excessive indulgence in sex causes Madhumegam.

Heredity :

“jhnd óUt éÂajdh‰ rhU«
Ãâf sšyhkš”
- njiua® thfl«

“ngWæsik æ‹g« Ãâ
_¥ò rh¡fhL MW§ fUéyik¥ò”
- nehŒ ehlš gFÂ 1

“R¡»y¤Âš Rnuhâj§fŸ fy§Fk‹W
ÓªÂL« éahÂ _‹W«”

- j‹tªÂç eho

“Kiw nf£»‹ x‹gJ Ka‰Áahš tªjJ
Jiw nf£»‹ f®¥g¤Âš Jt§»a nkf§nfŸ
eiu ó¤j bfh§ifahŸ ehaf‹ nkhf¤jhš
kiwngh‰W« f®¥g¤Âš ts®ªjjJ nkfnk”
- ÂU_y® fU¡»il it¤Âa« 600

“Â§fŸ g¤jh»š njŒbthL thÍ nr®ªJ
m§F ky®¤Â mkëah¡ Ñœ neh¡»
bgh§»¥ Ãwªj ehŸ òfœth® bgWÅz®
j§»¢ brå¡ifæš jh‹ nehÍ« c‰wnj”
- ÂU_y® fU¡»il it¤Âa«

Thirumoolar, Theryar and Dhavantri have noted Hereditary is one among the causes of Madhumegam.

Obesity:

“j‰gé¡Fª rßuªjh‹ äf¥g U¡fš
rŠryªjh‹ ga‹gLjš jç¡F« nehna”
- ô» it¤Âa ÁªjhkâÂ

“Ój Îzth‰ gjdêªj Ôid éU«Ã¤
Â‹gjdhš
XJ Kdé‹ nkYzth yl«ò äfÎ«
gU¤jydhš”
- guuhr nrfu«

Pararasasekarm says obesity is one of the main cause for Madhumegam.

Psychosomatic Cause :

“Ïa«gnt MWFs« Ã‹dŠ brŒjš
V‰wkhŒ Ãuhkz°Âß u§f« g©zš
ga«gnt ghy®fS¡ bfhë¤J Â‹dš
gHik ry« nghwtê jid¤ jL¤jš

ma«gnt Mya¤Â‰ ry«é£ nlh®¡F«
MÂah« ntj¤ij ¤öî‰ njh®¡F«
Ja«gnt Nçaid tz§fhjh®¡F«
RU¡fh nkf« tª J‰g é¡Fªjhnd”

- ôå Kåt®

According to Yogi Vaidya Chinthamani, Megarogam may occur due to not giving proper respect to Guru, Father, Mother, Vedas and Suriyan God.

MURKURIGUNAM [Premonitory Symptoms]

“jhfnk aÂf kh»¤ js®ªJ eh cy®ªjä¡f
nrhfkhŒ¡ »W»W¤J¤ bjhl®ªJif fhY«

- nrh®ªJ

VfkhŒ gfY« mšY« élhJ Úçw§F

- m‹¿

nkhfkhŒ kiH gå¡F KÂ®ªÂw§ »LŠ

- fyªjh‹”

- it¤Âa és¡f«

PREMONITORY SYMPTOMS

Premonitory symptoms of Madhumegam are polyuria, Polyphagia and Polydipsia.

Madhumegam exhibits the following premonitory symptoms from its initial stage of development itself. The patient experiences voracious hunger, thirst, perspiration, exhausion and giddiness. The excessive intake of water to quench his thirst is excreted as excessive quantity of urine.

- Noi Nadal Part - I

KURI KUNANGAL :

“j©ikahŒ¢ ryªjhD« gR¥ò kŠrŸ
jhåw§F« ÕrK§nfh rK§f L¡F«
m©ikah ao¡fo¡F Úç w§F«
mo¡fo¡F miuehê jåny fhQ«

bt©ikah aojå‰ wh‹ Ão¡F«
ä¡fd rl«btSªJ nkå f‹W«
g©ikahŒ gŠrth© gjå‰ bfhšY«
gf®»‹w kJnkf¥ gh§F jhnd”
- ô» it¤Âa Áªjhkâ

- Passing of urine in large quantity [650 ml each time] at frequency intervals.
- The urine is cold, slimy to touch, brownish yellow in colour and produce white sediments which adhere to the bottom of the vessel in which the urine is collected.
- The skin is pale and generalized tenderness all over the body and also feels dull pain in the testis and urethra during micturition.

nkf« bghJ F¿Fz«

“Twhd nkfkJ ÏUg J¡F«
Fzªj‹id¢ Át‹brhšy njénf£f
jhwhd jhfbkhL nrhf nkfª
jçahkš Úçêj èUkš _¢R
Mwhd mUÁr¤Â Á¤j¥ u«ik
mofo¡F¤ j©Ù®jh‹ M§nf nf£fš
<whd ÏL¥ò¡FŸ fL¥ò fhzš
vY«òH‰w yH‰wnyh blçÎ lhnk
vçnthL rßubkšyh kiwg£ lh‰nghš
vY«ò nehjš ã¤Âiuæš yhik
tçnthL khŒébk¤ jÎ«g ¿¤jš
kdJrŠ fy¥gLjš fh‰W nt©lhš
tçnthL khŒébk¤ jÎ«g ¿¤jš
kdJrŠ ry¥gLjš fh‰W nt©lhš
bkçnthL nkš_¢R äfÎ©lhjš
é¡fbyhL ka¡fªjh‹ bk¤j¡ fhzš

bjçnthL njfbk§F« btSU© lhjš

njfbk¤j thnyhg¥ gLjšfhnz”

- ô» it¤Âa Áªjhkâ

COMMON SYMPTOMS

Thirst	Polyuria
Polydipsia	Cough
Anorexia	Dyspnoea
Delirium	Pain in the hib and burning
Sensation	Loss of Weight, Body Pain
Sleeplessness	Flatulance
Hiccough	Giddiness
Anaemia	

NOI VAGAIKAL [Classification]

Megam is classified into twenty varieties.

To quote from Agasthir,

“c£oz nuhf¤jhY« cW«bgW« gÁædhY§
f£léœ nfhij khj® fyék£oyh ikayhW
K£lwh ehYkhW K«_‹W bkh‹W bk‹W
Â£lkhŒ tUtbj‹W ÂUKå aUë¢ brŒjh®”

- mf¤Âa® 1200

According to Therayar,

“fêÍ« thj« eh‹fhY« fhÍ« Ã¤j khwhY«
RêÍ« nr¤Jk« g¤jhY« brhšY« ehyŠrha
- njh‹W«

- njiua® thfl«

Dhanvantri’s Classification of Megam :

“ÃâbaD« nkfnuhf éÂæid¥ Ãç¡»š
thj«
gâ gæ¤Âa§ Ány‰g« ehyhW

g¤jhnk “

- j‹tªÂç it¤Âa«

Yugi Munivar Classifies the same as

“trå¤j nkfkJ æu©L g¤J
thj¤Â‰ Ãwªjy« ehfyahF«
Ãrå¤j Ã¤j¤ÂY ‰wgé¤j
nguhir yªjhD khW khF«
bjråªj nr£Lk¤ÂY‰g é¤j
Óuhd ryªjhD« g¤njahF«”

- ô» it¤Âa Áªjhkâ

Books	NOI	VALI	AZHL	IYAM
mf¤Âa® 1200	20	4	6	10
ô» it¤Âa Áªjhkâ	20	4	6	10
njiua® thfl«	20	4	6	10
j‹tªÂç it¤Âa«	20	4	6	10
rungªÂu ÚçêÎ nuhf Á»¢ir	20	4	6	10
ô»Kå it¤Âa fhéa«	2 0	4	6	10

The above books describe twenty different kinds of megam on the basis of colour, consistency, taste, smell, weight etc.,

Out of this twenty different kinds

Four varieties are caused by Vali

Six varieties are cuased by Azhal

Ten varieties are caused by Iyam

Madhumegam comes under the classification of Azhal. i.e. PITHA PRAMEHAM.

Classification of Megam :

According to Yugi Vaidhya Chinthamani.

thjÚ® tiffŸ :

jç¤Â£l thj¤Â‹ ryªjh dhY
å‚ahd ehY¡F« ngnu bj‹åš
mç¤Â£l M¢ÁabfªÂ nkf¤ njhL
mj‹ÃwF R‰wkh nkfbk‹W
Ãç¤Â£l Ãuäa nkf bkh‹W
nguhd kh§»rué nkf bk‹W

- ô» it¤Âa Áªjhkâ

Vali – 4

1. Aachiya Megam
2. Suthatha Megam
3. Pramiya Megam
4. Maangisa Megam

Ã¤jÚ® tiffŸ :

“Kiwahd Ã¤jry khW khF«
KÂªj m¥ Ãabk‹W« Ãuäa bk‹W«
jiwahd rh«Õ®zkJ«g bk‹W«
rh¤Âfnk ahWéjª j‹ndhlhW”

- ô» it¤Âa Áªjhkâ

Azhal – 6

1. Appiya Megam
2. Apiramiya Megm
3. Sambirna Megam
4. Madhumiya Megam
5. Saathiya Megam
6. Aavirutha Megam

IaÚ® tiffŸ :

“Mwhd Ány£gry« g¤J j‹id
mu‹brhšy M¤jhŸjh‹ nf£F«nghJ
thwhd trhnkf« cjfnkf«

k¢Áahnk f¤njhlh Ñj nkf«
öwhd Ruhç R¡y K¤j nkf«
R‰wkh«Ã dhåbahl yt© nkf«
nfwhd bjæ¤jŒkh nkf bk‹W
br¥Ãdh® Ány£g¤Â‹ brY¤J¤ jhnd”

IYAM :

1. Vasaa Megam
2. Utthama Megam
3. Maccha Megam
4. Aakhika Megam
5. Suraari Megam
6. Sukhila megam
7. Udaya megam
8. Pithathi megam
9. Lavana Megam
10. Thaithiya Megam

Yugi described four types under the vatha prameham, six types under the pitha prameham and ten types under kaba prameham.

“DIABETES MELLITUS” a clinical entity in a modern medicine is closely resembles one of the type of “Pitha Prameham” i.e. “MADHUMEGAM”

Ã¤j¤jhš njh‹W« nkfnehŒ M¿‹ bghJ F¿Fz§fŸ :

m¿ant Ã¤jry khW¡ Fªjh‹
m§fkÂ‰ brŒ»‹w Fz¤ij¡ nfshŒ
j¿ant rßu«t‰¿ baçÎ© lhF«
rl¤ÂYªjh ÜçYªjh‹ fé¢R©lhF«

bjçant Ó¥nghY§f‰ whiH nghY«

nršnghYª nj‹nghY eh‰w K©lh«

bt¿ant Õr¤Â‰nfh r¤Â‰ Fj¤Âš

äFÛuš ehÃæY« nt¡fh lhnk

- clš t‰¿ vç¢rY©lhF«
- clèY« ÁWÚçY« J®eh‰w« mo¡F«
- Û‹nghyÎ«, nj‹ nghyÎ«, eh‰wko¡F«
- Õr¤ÂY«, nfhr¤ÂY«, Fj¤ÂY«, <uš k‰W«
- ehÃæY« nt¡fhL c©lhF«.

Ã¤jÚ® tiffŸ :

1. m¥Ãa nkf«

RUtkh a¥Ãaªjh åw§F« nghJ

Joahid kj«nghy¤ jhå w§F«

btUtkhŒ ntis¡F ehdhê jhD«

éçrhf¤ jhåw§F§ fh¢Á d¡fhš

cUtkhÍ t®k©ngh‰ goÍ« ghU

c¡ukh¡ f©lbt£lh khj¤ j‹åš

Ïutkh bakòu¤J¡ bfŒJ thuf‹

X‰wkh k¥Ãa¤Â‹ nkfª jhnd

- ÁWÚ® fê¡F«nghJ mjDl‹ ahid kj«nghy Ïw§F«
- XU ntis¡F eh‹F ehê Ïw§F«
- mªj ÁWÚiu fhŒ¢Ádhš ct®k©nghš goÍ«

- ÏªnehŒ f©l v£lhtJ khj¤Âš nehahë Ïw¥g®

2. mÃu«äa nkf«

“jhbd‹w f‰whHŠ rhW nghY«
j‹dh‰w« nghynt äfnt ehD«
fhbd‹w fh¢Ádh‰ foj eh‰w«
foif¡ FëUehê ju w§F«
Vbd‹w é¡Fzkh nkfª jhD«
Ïw§»dnjh® _‹whF kh©L j‹åš
ghbd‹w gukgj bkŒJ bk‹W
ghkDiu mÃu«äa¥ g©ò jhnd”

- ÁWÚ® Ïw§F«nghJ f‰whHŠrhW nghy ÏU¡F«
- J®eh‰w« mo¡F«
- fhŒ¢Ádhš mÂf J®eh‰wko¡F«
- xU ehêif¡FŸ xU ehêasÎ ÁWÚ® fêÍ«
- ÏªnehŒ f©lt®fŸ _‹wh©Lfëš kuz« vŒJt®.

3. rh«Õ®z nkf«

“g©ghf¢ R©zh«ò mHny ehW«
goÍ« aojåny ÚW nghy
e©ghf ntis¡F ehdhê ehD«
eh¿na æw§Fäf btU« gç¡F«
f©gh®¡ f¡fh¢Ádh‰ R©z ÚU«
fdkhf goÍnk aoæ‰wh D«
j‹ghf¡ f©lry äu©lh kh©o‰
rhFtnu rh«Ã®z¤ j‹ik jhnd”

F¿Fz§fŸ

- ÁWÚ® R©zh«ò j©Ùiu¥ nghy eh‰wbkL¡F«.
- moæš g‰g«nghy bt©ikah» goÍ«.
- xU ntis¡F eh‹F ehê ÁWÚ® Ïw§F«.
- vW«òfŸ bkhŒ¡F«. ÁWÚiu¡ fhŒ¢Ádhš R©zh«ò goÍ«.
- Ïªnehœ f©l Ïu©lhtJ M©oš kuzbkŒJth®fŸ.

4. kJäa nkf«

“j‹ikahŒ¢ ryªjhD« gR¥ò kŠrŸ
jhåw§F ÕrK§nfh rK§f L¡F«
m©ikah ao¡fo¡» Úçw§F«
mo¡fo¡F miuehê jåny fhQ«
bt©ikah aojåš jh‹ Ão¡F«
ä¡fhd rl«btS¤J nkå f©Q«
g©ikghŒ¥ gŠrt®z gjå‰ bfhšY«
gf®»‹w kJäa¤Â‹ gh§F jhnd”

F¿Fz§fŸ

- Ú® gRkŠrshf¢ bršY«
- ÕrK«, nfhrK« fL¡F«
- mo¡fo Úçw§F« x›bthU KiwÍ« miu ehêasÎ ÁWÚ®¡ fêÍ«,
- ÁW Úçš moæš bt©ikahf¥ goÍ«
- clš btS¤J nkå f‹åéL«
- IªJ M©Lfëš, mªnehahë kuzkilth‹

5. rh¤Âa nkf«

“gh§fhd gë§Fãw« nghè w§F«
gçÕr nfhrKnk fL¤J¡ TR«
jh§fhd jhiHbahL éGJ rhW

jid¥nghy¤ jhå w§F« rh¢Á d¡fhš

nj§fhd Ó¥nghy äfnt ehW«

Ó¡»ukh ntis¡nfh® gojh dhF«

V§fhd Ï¥go¤jh‹ f©l nkf«

vŒÂLnk Iªjh©oš mrh¤Âaª jhnd”

F¿Fz§fŸ

- ÁWÚ® gë§Fãw« nghyéU¡F«
- ÕrK«, nfhrK« fL¤J¡ TR«
- jhiHéGJ¢rhW nghy ÁWÚ® Ïw§F«
- Óœnghy äfÎ« ehW«.
- XU ntis¡F xU goasÎ ÁWÚ® Ïw§F«
- Iªjh©oš kuz« neçL«.

6. MéUj nkf«

rh¤ÂakhŒ Kršu¤j« nghy ÅG«

rhjfkh æUehê jhå w§F«

fh¤ÂakhŒ¡ fh¢Ádh‰ òyhny ehW«

fdkhd Õrnfhr§ fL¡ Fnk

C¤Âahkh æ¥gojh‹ f©l nkf«

X‹gjh khj¤Âbyh L¡fŠ Ót‹

M¤Âakh khéUj nkf bk‹nw

MtåÍŸnsh® j§fS¡ f¿a¢ brhšny”

F¿Fz§fŸ

- Kaš Ïu¤j«nghy ÁWÚ® fêÍ«.
- XU ntis¡F Ïu©L ehê Ïw§F«.
- ÁWÚiu¡ fhŒ¢Ádhš òyhš eh‰w« vL¡F« ÕrK«, nfhrK« fL¡F«.
- Ïªnehœ f©lhš x‹gjh« khj¤Âš ÏwªJéL®.

mDgt it¤Âa njt ÏufÁa« g¡f« 131

nkf nehŒ :

kJnkf«, k¢rh nkf«, trh nkf«, m°Â nkf« vd eh‹F« thj¤Âdhš c©lhF«.

kŠÁZo nkf«, rhu nkf«, mç¤Âuh nkf«, Úynkf«, u¤j nkf«, fhs nkf«, v‹D« Ï›thW« Ã¤j¤Âdhš c©lhF«.

ÕZl nkf«, Ï£R nkf«, rhªÂuh nkf«, Áfjh nkf«, yhyh nkf«, Ój nkf«, rdd® nkf«, Ruh nkf«, r¡»y nkf«, ry nkf« v‹D« g¤J« fg¤Âdhš c©lhF«.

j‹tªÂç it¤Âa« - Ïu©lh« ghf« g¡f« 298

kJka nkf¤Â‹ Fz« :

“kJka nkfkhdhš tªÂL§ Fz¤ij¡ nfshŒ
gÂÎw¤ j©ÙU©Q« gh®¡»š Ú® òyhny - ehW«
éjbgw¡ fhŒ¢Á¡ f‰f« éshéa nj‹ - nghyhF«
òÂa bkš yä®jé‹ brh‰ óitna - a¿Fthna”

Symptoms :

- Increase of thirst
- Polydypsia
- Fatty smell in urine
- On heating urine becomes honey like consistency

mf¤Âa® MÍŸ ntj« - 1200 g¡f« 243

1. br¥òkh§fyaª j‹å‰ Áw¥òl‹ ry¤ij - it¤jhš
x¥òl‹wæU« nghy ÎiuªJ jh‹ ÑnH ã‰F
bkŒ¥òl‹rßu« t‰¿äFªjÂl¤jhf K©lhf
k¥bghGJjrdŠ bršyhja‹¿L §ifÍ§fhny

Symptoms :

- Urine settles like curd
- Emaciation of body

- Polydypsia
- Anorexia
- Weakness of extremitites

2. “iffhya®ªj beŠRy®ªJ fd¤j btçÎ
bkŒjh‹¿wªJ eil bfhŸsh ÛSäj‰F¥ - K©lhF
- gçfhuŠ”

Symptoms :

- Burring sensation of the body
- Inability to walk

nkf ãthuz nghÂå vD« ÚçêÎ kU¤Jt« g¡f« 150

njf¤Â‹ kJu¤ij cU¡»¡ bfh©L tUtjhš, kJnkf« vd¥ bga® bg‰wJ. ÏJ

1. Éuz kJnkf«
2. óuz kJnkf« - vd ÏUtif¥gL«

Óuz kJnkf«

ÏJ K‹T¿a ó®t%g§fis milªJ nuh»ia btF Ó¡»u¤Âš bfL¡F«. Mifahš Ïj‰F Óuz kJnkf« vd¥ bga®. ÏJ mrh¤Âank.

óuz kJnkf«

ÏJ K‹T¿a ó®t%g§fis milªJ nuh»ia mÂÓ¡»u« bfL¡fhkš beLehis¡F it¤ÂU¥gJ« c©L. Mifahš mj‰F¥ óuz kJnkf« vd¥ bga®. ÏJ fZlrh¤Âank.

MUKKUTRA VERUPADUGAL [PATHOLOGY]

The disease madhumegam due to external or internal causes affect balance in the ratio of Vali, Azhal, Iyam. The imbalance affects the Keelnokkukal which inturn affect the seven udal thathukkal. Saram gets affected and there is loss of appetite. Seeneer also get affected with the net

result even if the patient eats more nourished food [polyphagia], there wont be any improvement in health.

An imbalance in Iyam does imply an imbalance in other two kutrams too and causes derangement of dasavayu and seven udal thathukkal which cause the disease and other complications.

"F¿Ílnd nkfªjh‹ bfhLik brŒJ
FiwªJ tªj tUªjhJ btšyh§ - F‹¿¥ngh«"
- gÂbz‹Á¤j® eho üš

PINIYARIYUM MURAI – [DIAGNOSIS]

This is the method of diagnosing the disease. It is based upon three main principles.

1. Poriyal Arithal
2. Pulanaal Arithal
3. Vinaathal

Pori is considered as the five senses of perception namely Nose, Tongue, Eye, Ear and Skin. While pulan are five object of senses. They are smell, Taste,Vision, Sound and sensation. Pori and pulan are used as the tools for examining the patients.

Vinaathal is obtaining the informations regarding the history of the disease, clinical features etc. from the patient or their relatives.

When the patient is unable to speak or if the patient is a child.

Poriyal Arithal and Pulanaal Arithal and Vinaathal are used to diagnose the patients. In modern medicine interogation, inspection, palpation and percussion are used to clinically diagnose the disease.

ALAVAI

Alavai is also useful for diagnosing the disease. Alaval is a parameter through which one can asscess the real properties, merits and demerits of things using the five sense organs as the instruments.

Among the ten classification of Alavai the first three are very important and helpful in the examination of a patient. It is useful in diagnosing Madhumeham as follows :

Kaandal Alavai - [Observation]

By observing the attraction of ants and flies to the vaided urine. We can persume that the urine is sweet in taste.

Karuthal Alavai – [Inference]

When the patients complaints of Polyuria, Polydipsia, Polyphagia, loss of weight etc. the physician can have a clue to the diagnosis of madumegam.

Uri Alavai : -[Authority]

"ÏUäna Ã¤jK« thj« Toš
kUÎ rynkf« thUÂ nghyhF«
cUt« ntwhFK©l Îl‰ fhªÂL«
cUfnt ñndhL c¿ŠÁ Ïå¡Fnk"
- ÂU_y®

Thirumoolar says that pitha vatham naadi felt in the madhumegam patients for further confirmation of the disease.

Thirumoolar describes the excretion of sugar in the urine in due to the combined vitiation of pitham and vatham. Poriyaal arithal, Pulanaal arithal and vinaathal are effected through eight types of investigations. That is envagai thervugal.

ENVAGAI THERVUGAL :

Naadi

Sparisam

Naa

Niram

Mozhi

Vizhi

Malam

Moothiram

“eho °gçr« eh ãw« bkhêtê
ky« _¤Âu« Ïit kU¤JtuhÍj«”
- nju‹

Sparisam – [Palpation]

The following prints are elicited by sparisam temperature of the skin, any abnormal growth hyper – sensitiveness, thickening of the skin, swelling, ulcers etc.,

In Madhumegam dry skin and peripheral neuritis present.

Naa – [Tongue]

The colour, dryness, excessive salivation, coated or not, redness ulceration, pallor, yellow discoloration, any growths, condition of teeth and gums are noticed. Taste, movement of tongue and speech are also made out.

In Madhumegam dryness of the tongue.

Niram – [Colour of the skin]

Colour indicating vatham, pitham, kabam or kalappu udal, yellow or pallor or redness or bluish discoloration may occur in pathological conditions.

Mozhi – [Voice]

Any speech disturbances, loud voice, slurring, crying, talk induced hallucination undue argument, breathlessness can be made out.

In Madhumegam Mozhi is not affected.

Vizhi – [Eyes]

Any abnormal colour changes indicates derangement of mukkutram. Pallor, excessive lacrimation, sub conjunctival haemorrhage, falling of eye brows and visual disturbances are made out.

In Madhumegam Diabetic retinopathy present.

Malam – [Faeces]

Quantity, colour, odour, abnormal consistency, Froth and frequency or constipation are made out.

In Madhumegam constipation present.

Moothiram – [Urine]

Quantity, colour, odour, frothy, frequency, retantion, deposits heaviness and presence of abnormal constituents such as sugar and neerkuri and neikkuri are made out.

In moothiram poly uria present.

Naadi – [Pulse]

“cæ®¡fhjhu« Kæ®¤jh bjdÎ«
K¥Ãçth» K¡Fz kQ»
cliyÍ KæiuÍ nkh«Ã¡ fh¤J
tUbkd KJkiw tF¡Fª Jâng”

Naadi is omnipresent cosmic vibrant force connecting the macrocosmic with the human body is a subtle diagnostic tool handed by the Siddhars. These vibrations enter into the human body from the universe, keep the life vibration

continuously and generate the energy required for the human Metabolism. Naadi is the vital force. The examination of naadi has been recognized as one of the principal means of diagnosis and prognosis of the disease from time immemorial. Any change in the mukkutham is best diagnosed by feeling the naadi. The power of Naadi manifests in the body as three vital forces namely vatham, pitham and kabam. The three uyirthaukkal which organize, regularise and integrate the life activities in each and every living being.

The same three fundamentals are described as Edakalai, Pinkalai and suzhimunal in yoga texts which describes the methods for preventing disease and for prolonging life to attain wisdom and salvation. The same explanation is also in Siddha texts for understanding.

The unity in diversity to achieve health, wealth and happiness and satisfaction.

On the basis of the examination of the senses and on the basis of eight special examination and interogation, all the details of the disease factor are collected and their final diagnosis is confirmed with those findings made in Naadi parichai.

NAADI NADAI

“ÏUäna Ã¤jK« thjK« Toš
kUÎy nkf« thUÂ nghyhF«
cUt« ntwhF K©lÎl‰ fhŒªÂL«
cUfnt ñndhL c¿ŠÁ Ïå¡F«
gh®¤ÂL _‹W« gÂªJ bkèªJ ã‰»š
nj®ªÂL nkf« cŸns njh‹¿na bghUªÂbkŒæš”

- ÂU_y® eho

The above poem says that excessive elimination of urine containing sugar are always primarily due to combined vitiation of pitham and vatham.

Functional factors in the body, the vitiation of pitha vatham is indicated clinically by excessive hunger, thirst, emaciation and passing of large quantities of urine.

“Ïå¡»‹w thj¤ Âilnrçš Iaªjh‹
gå¡»‹w fŸS« gjånghš ÚnuhL«
få¡»‹w nkå fiuªJ btS¥ngW«
jå¡F« kJnkf« j¥ghnj IaK«”

- ÂU_y® eho

According to the above stanza initially vatham and kabam are deranged, then it leads to vitiation of pitham finally. When the vitiation vatham combines with vitiation kabam there is genesis of mega noi in the body. Thus urine has consistency and appearance of toddy or sweet toddy and the body is emaciated and pale. This is the typical clinical picture of madhumegam.

“JuzKl‹ Ú®ghL ne®gh lhdhš
brhšY»‹nw‹ ehobašyh« js®ªJ fhQ«”

- gçóuz eho

From the above lines, all the three naadies are feeble and weak in madhumegam patients.

“g‰Ãlªj nkfbk‹whš Ã¤j ÛS«
ghyfnd fh§if bfh©L ãuh« ghnu”

From the above lines it is clearly stated that vitiation of pitham results in madhumegam.

“Ú®nkf khdt®¡F eho jhD«
Ú®kakhŒ ehobašyh« gynk bf£L¡
fh®nkf« nghynt tª bjçnkš òu©L
éG¥òG¥ nghynt òu©L fh£L«”

- gçóuz eho

All the three naadies are felt feeble in those suffering from mega noi. The character of the pulse is compared to that of wriggling movements of a worm that has fallen into the fire.

“cWÂÍŸs Ã¤jkJ njh‹¿š
... ... Ãu nkf§fŸ”
- rjf eho

“Mnkjh‹ a¤Â Ru«gh©L nrhif
MHhd élh¢ RuK« Ãunkfªjh‹”
- - -Ã¤j¡TW

eé‹¿£lh® thrKå eé‹ ¿£lhnu
- mf¤Âa® eho

The above stanzas are indicating vitiation of pitham, resulting madhumegam.

“Áw¥ghd thj¤Â Y£oz‰ jhnd
- - -
Ãunkf« - - -
Mdgy ÃâfSnk tªjlUª jhnd”
- rjf eho

The above stanza indicates that the vatham combined with heat manifests madumegam.

MUKKUTRAM:

Vali, Azhal and Iyam

The description is anatomical and physiological. Vatham has been described in ten forms with various functions pitham and kabam have been classified in five forms with different functions.

Location of Vali in the body :

Vali is located in the abaanam, Urinary bladder, intestine, idakalai, spermatic cord, pelvic area, skin, nerve endings, joints, hair and muscles.

Location of Azhal in the body :

Azhal is located in piraanavaya, bladder, moolakini, heart, Umbilical area, abdomen, sweat, saliva, blood, eyes and skin.

Location of Iyam in the body :

Iyam is located in Samaanavayu, Sperm, head, tongue, vulva, fat, bone marrow, nose, chest, nerves, brain, eyes and joints.

VALI – PIRIVUGAL

1. Piraanan - [Uyirkkaal]

This controls knowledge, mind and objects of five sense organs and useful for breathing and digestion.

In diabetic keto acidosis, kussmaul's air hunger with hissing respiration occur due to affected piraanan.

2. Abaanan – [Keezhnokkukaal]

Responsible for all downward movements such as passing of urine, stool, sperm, menstrual flow and constriction of anal sptincter muscle.

Polyuria, constitpation or nocturnal diarrhoea due to affected abaanan.

3. Uthaanan – [Melnokkukaal]

Responsible for all upward visceral movements such as vomitting, eructation and nausea due to autonomic or visceral neuropathy, excessive thirst.

4. Viyaanan – [Paravukaal]

Responsible for movements of all parts of the body and sensation. This is due to symmetrical sensory polyneuropathy, pain all over the body. Burring sensation in the sole of foot and palm, skin infection and carbuncle.

5. Samaanan – [Nadukkaal]

Responsible for digestion and absorption of food and water. Polyphagia due to affected Samaanan.

6. Naagan :

Responsible for knowledge, opening and closure of the eye, Affected III and VI cranial nerve also seen in mononeuropathy. Diminished vision due to Naagan.

7. Koorman :

Responsible for vision and yawning. Diabetic retinopathy and cataract are common in diabetes.

8. Kirukaran :

Responsible for salivation, nasal secretion and appetite.

9. Devadathan :

Responsible for lazziness, sleep and anger. In madhumegam – normal

10. Dhanancheyan :

Produce bloating of the body after death. It escapes on the third day after death by bursting the skull.

AZHAL

Sites of Pitha :

Between the heart and the naval, sweat, lymph, blood, stomach, urinary bladder, heart, saliva, eye and skin properties.

Dry, cold, hot, light, subtle, keen, soft, liquid, bitter

Functions :

Body temperature, digestion of food colouring of the skin, vision, sweat

In Madhumegam

Analapitham : Excess Hunger

Ranjaga Pitham	:	Pallor Sometimes
Alosaga Pitham	:	Diminishing of Vision
Saathaga Pitham	:	Lassitude
Pirasaga Pitham	:	Dry Skin

IYAM :

Sites of Kapha :

Above the heart, stomach, fat, sperm, tongue, uvula, borne marrow, blood, nose, nerves, bones, large intestine, eyes, joints.

Properties :

Heavy, cold, Mild, Watery, Sweet, Stable and Slimmy.

Functions :

Kapha gives strength builds the body gives strength for joints, gives shiny appearance to the skin, moistens food, cools the eyes, gives a whitish colour to the conjuctiva, skin, urine and faecal matter, softness, firmness, the capacity to bear or endure, patience.

Abnormal functions :

Whiteness of complexion, cold, itching, dullness, heaviness, oilyness, loss of sensation, tightness as if born with corns, a sense of sweetness in mouth, procrastination in respect of work.

Avalambagam :

Affected due to effect of other varieties of kapha.

Kilethegam :

Excessive appetite

Pothagam :

Dryness of the tongue

Tharpagam :

Burning sensation in the eyes.

Santhigam

Joint pain.

Ú®ãw¡F¿ :

“mUªJkh¿ujK« ménuhjkjhŒ
m~fš my®jš mfhyñ‹ jé®ªjH‰
F‰wstUªÂ cw§» itfiw
Mo¡fyr¤ jhéna fhJbgŒ
bjhUKT®¤j¡ fiy¡F£gL Úç‹
ãw¡F¿ beŒ¡F¿ ãUä¤jš flnd”

Collection of Sample Urine :

The patients must take well cooked food in the previous day. The intake must be proportionate time. We must have sound sleep on the previous night. The urine is collected on the dawn of the next day in a glass container and closed immediately to prevent contamination. This specimen must be examined within one and half hours. This procedure should be followed strictly to get accurate observation of neerkuri and Neikuri.

beŒF¿ :

“ãw¡F¿¡ Fiu¤j ãUkhz Úç‰
Áw¡f bt©bzŒnah® ÁWJë eLéL¤
bj‹Ww¤ Âwªbjhè nafh jik¤jÂ
å‹wÂtiy ngh« be¿éêa¿Î«
br‹wJ òfYª brŒÂia Íznu”

NEIKURI

The diagnosis and prognosis of deranged MUKKUTRAMS are studied on the basis of the behaviour of a drop of gingelly oil gently dropped on the surface of the urine kept in a wide vessel in the sunlight.

“K¤bjh¤J ã‰»‹ bkhêtbj‹ fgnk”

In Iyya Madumegam, the oil dropped in Urine is like a pearl and if the oil spreads slowly, the prognosis of the disease is slow and good.

Úçêî nehæš fhQ« mt¤ijfŸ :

The following complication follow gradually if the disease is not controlled of left untreated.

mt¤ij 1 :

“fhznt Kjyt¤ij¢ rßuª jhD«
fdkhf¥ gU¤ÂWF Ú®¤J thu«
ntznt bt©lh¡» afy« g©Q«”

- Obesity sets in
- There is obstruction in urinary flow urinary passage expands due to inflammation

.

mt¤ij 2

ä¡f Ïu©L lhkt¤ij és«g¡ nfshŒ
_znt _¤Âu¤ ÔilÍkh¢ R¡y
KfkH»¤ nj#Rjh‹ äfnt F‹W«

- Micturation is frequent
- Sexual desire gets reduced
- There is also loss of complexion

mt¤ij 3

“ehznt _‹whF kt¤ij¡ Fªjh‹
ehtwS« thÍtJ ÛWª jhnd”

- Tongue generally becomes dry
- Abdomen is distended due to flatulence

mt¤ij 4

“jhdhd ehyt¤ij a§f jhf«
r‹åaJ ghjK©lh«”

- Severe thirst occurs
- Causes delirium

mt¤ij 5

“lªJ t¤ij¤
njwhd Ú®¥ bgUFª jhJ eZl«”

- Quantity of urine increased
- Loss of semen (Impotance)

mt¤ij 6

“ãiy ahwh kt¤ijÍl‰ »ilbfhŸ shJ
_dhd _®¢ir tU«”

- Sleeplessness is present
- Difficulty in breathing is experienced

mt¤ij 7

“VHt¤ij
ä¡ftnuh ÁfŠRthrª njr rh£a«”

- Tongue becomes tasteless
- Difficulty in breathing is experienced
- General Weakness persists.

mt¤ij 8

“Vdhd v£lht jt¤ij jhnd
vG»uªÂ ÃsitÍªjh‹ äfÎ© lhnk”

- Abscess is formed
- Presence of carbuncle

mt¤ij 9

“c©lhF bkh‹gjh kt¤ij nfshŒ
xG§fhd Mrhuh§ »Uä Í©lh«”

- Irregularities in daily habits like bowel habits.
- Bed soar may occur

mt¤ij 10

“g©lhd g¤jhªj it¤ij¡ nfshŒ
ghukh« ra§f©L gu¤J¡nfF«”

Secondary infection like tuberculosis may set in due to loss of immunity other complications leading to death may occur.

These are the complication of 20 Madhumegam.

Other Complications :

Meganeeer Kattigal 10

1. Madaku Katti
2. Ammaiodu Katti
3. Valai Kaan Katti
4. Athomuga Katti
5. Phai Sunai Katti
6. kadalai katti
7. Kadugu Katti
8. Vithirathi Katti
9. Nilapposani katti
10. Magavithirathi Katti

ÔU« Ôuhjit :[Prognosis]

“brŒant t¢ru khª j©l khd
brakhd KJFj©il¥ g‰¿ ã‰F«
bgŒant bgUeu«Ãš nkfª jhD«
Ãw¡Fbk‹nw jhd¿ªJ thjª j‹dhš
ÃŒant Ãwªjfy kheh yrh¤Âa«
Ã¤j¤Â‰ Ãwªjry khW« ah¥a«
igant nr£Lk¤Â‰ Ãwªj g¤J«
gukDiu¤ jh® rh¤a« guhgç¡nf”

- ô» it¤Âa Áªjhkâ

“MdJ Ã¤jª j‹å yhwŠ nr‰gd« g¤
Ôdkh« thj eh‹fh äÂš thj® Ôuh - Únu”
- guuhr nrfu«

“têÍ« thj« eh‹fhnk khwh jéœjª - j‹dhny
bghêÍ« thj« ãšyhJ nghnk kUªij¥ - bghŒbadnt”
- njiua® thfl«

The four types of megam caused as a result of imbalance of vali are incurable.

The six types of megam arising with disparity of Azal could be cured with great difficulty.

But ten types of megam arising due to Iyyam are curable.

vªbjªj nuhf§fëš ÁWÚ® mÂfç¤jhY« FiwªjhY« ÔJ ?

“bt¥ò Ãâajåš bt«nkf¤jhš tUªÂ‹
j¥ò äif Únu jhåw§»‹ - br¥ò«
»uhâæ‰ gh©oš »s®Ú® RU§»‰
Ãuhz‹ ÃçÍbkd¥ ngR”
- bt©gh - f©Qrhäa«

Very excess of urination in Madhumegam cause death.

“JÂ¥ghd nkf¤Âš ÚçêÎ khfh
njh‹¿a ÚçÆÎ j©Ù® thjKkhfh “
- rjf eho

If Megaroham is associated with excessive urination, it is difficult to cure. If Madhumegam coexist with vali it is incurable.

“nkf¤Âš ÚçêÎ nkÎkÂš thjnehŒ
ntf thj¤JŸ tæ‰WisÎ - nrhf é¡fš
g‹D é¡fš j‹åš gfçis¥ò¥ gh§fjå‰
Ã‹dis ahfhJ ngR”

In Megaroham Valinoi, excurating pain in abdomen, hiccough and tuberculosis coexist it is incurable.

“Úç‰ Ãsit ãfœÃsitæš jhf«
XJ kÂš njf¤JW kdYª - njUkÂš
nrhU ka¡fKŠ brhš ka¡f¤Âš éa®it
Mç‰Öjh bk‹w¿”

If complications of madhumegam with carbuncle, morbid thirst, excessive body heat, shock and sweat occurs the prognosis is bad.

MARUTHUVAM

it¤Âa¢ braš it¤Âakhnk
- ÂU_y® 8000

In Siddha System, treatment is not only the removal of disease, bt for the prevention and improving the body condition.

Kappu - Preveniton

Neekkam - Treatment

Niralvu - Restoration

KAPPU :

Prevention is better than cure is a proverb. Siddha Principles based mainly on prevention as mentioned in “Therayar Pini Annuga Vithi” by Therayar. Siddhars have proficiency in astrology and by looking at the allignment of twenty seven stars and nine planetary arrangements, the disease whether curable or not can be easily identified.

NEEKAM :

The aim of the treatment is to bring the affected thathus to normal levels by Eyamma, Niyama Diet and Medicine.

For the disease Madhumegam, Sarabanthra Madhumega chooranam 1 gm thrice with hot water after food and Madhumega kudineer – 30 ml twice daily before food is given.

NIRAIVU :

Physical, psychological, Social and economic rehabilitation of individual is known as Niraivu.

In Madhumegam Azal Kutram and other two kutrams Azhal and Iyam deranged and causes impairment of dasavagy, which in turn affect the seven udal.

LINE OF TREATMENT

"gf®Ã¤j éªijayhJ nkf« tuhJ"
- njiua®

So treatment must be done to correct Azhal and then to correct other two kutram Iyam and vali.

KUDINEER

Avvarai Kudineer 100 ml with butter milk. Thetran seed, Kadugai Mesoderm, Avvarai seed, Vilam pesin in equal quantities with butter milk. Commonly used choornam are

Tripala Chooranam
Navalkottai Choornam
Madhumega Choornam
Sirukurinjan Choornam

} Any drug 1 gm 3 times a day.

Abiraga parpam 60 mg thrice daily. [or] Abiraga chendooram 60 mg thrice daily also be used.

In therayar maruthuva Bharatham the root disease Megha is compared to Kisagan and Madhumegam is compared to Sayanthevan.

Excess indulgence in sex causes Megam that is kisagan who has less mortality is killed by Bhiman who is compared to Mercury. So far Megam Mercury compounds with sulphur is given.

Sayanthavan was killed by Arjunan. The disease Ayya Madhumegam is treated with copper compounds sempu parpam.

To strengthen seven udal thathukkal saram is improved by giving rasa and Gandhaga drugs

Senner is regulated by giving Iron containing drugs [Haematonic] which is compared to Dharman.

Iron is strengthened by velli which prevent lipolysis, compound to sagadavan. Kozhuppu and Ennpu are strengthened by Gold Preparation which is compared to Abimanyu.

Moolai and sukilam is strengthened by Kariyam and sembbu which is compared to Krishna and Arjuna.

So pancha pandavas are alluded to metals which are given for treating Madhumegam. Generally these metals with sulphur is used for treatment is more and has good efficacy which is based on Vinthu Natha Principle.

Nutritional Approach :

The approach consists of provision of adequate calories for maintaining or attaining standard weight for age, sex and height.

General conseasus on proportion of food constituents are

Carbohydrate	-	60 – 65%	[Complex Form have more Fibre]
Fats	-	20 – 25%	[Saturated 7.8%]
Poly unsaturated	-	7 – 8%	[Mono unsaturated 7.7%
Proteins	-	10 – 15%	

Disease like Madhumegam has very important role in diet and Nutrition Diseases are largely due to irregular dietary habits. The body requires no medicine no if new food is eaten only after the food that has already eaten before is fully digested and the food agrees with body.

To quote from Thirukural,

“kUªbjd nt©lhth« ah¡if¡

fUªÂa j‰wJ ngh‰¿Í©â‹”
“m‰wh yst¿ª J©f m~Jl«ò
bg‰wh‹ beLJa¡F khW”

Proper diet according to the digestive fire, age of the individual prevents illness says Thiruvalluvar.

To live healthy, specific food are mentioned for six different seasons.

Seasons	Food
Karkaalam Munpani Kaalam Pinpanikaalam	Warm Food with Sweet Sour, Salt Taste
Kuthirkaalam	Sweet, Bitter, Astringent foods which are dry
Elavenil Kaalam	Food with pungent, astringent, bitter taste which are warm
Mudhuvenil Kaalam	Sweet and Cold Foods

nkf« ÏUgJ¡F« g¤Âa« :

“gRnkf äUgJ¡F g¤Â aªjh‹
gh§fhf Îiu¤Âlnt gRé‹ bt©bzŒ
bghçthf vUiknkh® bgh‹dh§ fhâ
ng®bg‰w ÁWÑiu KR£il ahF«
mçthf mtiubahL òlšK U§if
mÂrukh§ f©LrU¡ fiuÍ khF«
kçthf khjisah« ngß¤ jhF«
kfhés« gHKªÂç¥ gHK khnk
Mnkng a‹thiH¥ gHK§ f¢rš
m¤Âæl¥ ÃŠRÁW gaWgHŠ nrhW
ghnkgHŠ nrh‰WÚ® btªjaŠÓ ufkh«
ghfšÕç¡ f§fhŒfU nt¥Ãiybfh¤jkè
nebkbe‰ bgh¿vŸS KRKR¡if ahF«
neuhd ešby©bzŒ ò©z¡ FSªJ
jhnkf äUgJ¡F« g¤a t®¡f«
rh‰¿dh® Át‹whDª jhŒ¡Fª jhnd”

Ñiu tiffŸ	fhŒf¿fŸ
bgh‹dh§fhâ	mtiu

ÁWÑiu	òzš KU§if
KR£il	thiH f¢rš
bfh¤Jkšè	m¤Â¥ ÃŠR
fUnt¥Ãiy	ghfš
KRKR¡if	Õ®¡F

fåfŸ	**bfhG¥ò bghU£fŸ**	**éij**
khJis	gRé‹ bt©bzŒ	bt^{a}ja«
ngß^{a}J	vUik nkh®	Óuf«
ésh«gH«	ešby©bzŒ	vŸ
K^{a}Âç¥gH«		
nga‹ thiH		

khÎ bghUŸ	**gæW tiffŸ**
gHŠnrhW	ÁW¥gæW
gHŠnrhW Ú®	cS^{a}J
be‰bgh¿	

Madhumegam patients are advised to use lot of greens, pulses, vegetables, fruits and seeds. These foods contains lots of fibro, which has the property of holding water and swells and behaves like a sponge as it passes through the gastro intestinal tract.

Fruits are a very good source of several vitamins, minerals and fibers. They condain fructose and other sugar depanding on the sweetness. The fruits mentioned above are less sweet in taste.

The few fat substance they use have curative nature against madhumegam that is.

Cows butter cures pramegam

Buggaloe’s butter milk cures thirst

Ginglly oil cures disease of eyes, ear, kin infection like scabies, ulcer etc.,

Most of the above food used for Madhumegam are “Sathva Gunam”.

This clearly implies in Madhumegam patients the stress level is very high and to reduce that “stress” improve tolerence of disease sathva guna foods are used.

YOGA:

Yoga is the most valuable inheritance of the present. It is the essential need of today and the culture of tomorrow.

- ***Swami Satyananda Saraswathi***

Yoga physical exercise makes the muscles healthy and strong. It also tones up all the involuntary organs of the body which are concerned with the processes as digestion, evacation, circulation, respiration and secretion and through them, the autonomic nervous system which regulates their activities.

- **Yogic Asanas for Health & Vigour**

“Ïak ãaknk v©âyh Mjd«
eaKW Ãuzhahk« Ãu¤Âahfhu«
raKW jhuiz Âahd« rkhÂ
maKW m£lh§ khtJ khnk”

- ÂU _y®

Each and every yogaasanam is indicated for a definite effect in aparticular region of the body, by stimulating the internal organs to function in a normal way.

The following assanaas are advised for Madhumegam.

1. jDuhrd«
2. g£Ánkhjhrd«
3. m®¤j k£rnrªÂuhrd«
4. Ayhrd«

ANATOMY

The Pancreas

Situation:

- Pancreas is a fleshy gland with endocrine and exocrine functions.
- It extents upto the hilum of the spleen
- It lies on the posterior abdominal wall.
- It lies below the liver
- It lies deep to the stomach.

Length :

14 – 15 cm

Weight :

80 to 90 grams

Parts of the pancreas :

Head, reck body and tail.

Head :

The head of the pancreas lies within the concavity of the duodenum.

It lies on

a. Inferior vena cava
b. Renal veins
c. Portal vein
d. Bile duct

Body

The body of the pancreas lies on the

a. Abdominal aorta
b. Left kidney
c. Left supra renal gland

Tail :

The tail of the pancreas lies on the Hilum of the spleen.

Pancreatic duct :

1. Major pancreatic duct [Duct of Wirsung]
2. Accessory pancreatic duct.

The major pancreatic duct unites with the bile duct and forms hepato pancreatic ampulla. It opens into the second part of duodenum.

The accessory pancreatic duct is present in the head of the pancreas. It also opens into the duodenum.

Blood Supply :

1. Superior pancreatic duodenal artery
2. Inferior pancreatic duodenal artery
3. Splenic artery

Venous drainage :

1. Splenic vein
2. Superior mesenteric vein
3. Portal vein.

Nerve Supply :

Para Sympathetic - Vagus nerves

Sympathetic - Coeliac plexus

Structure of the Pancreas :

- It is made up of lobes and lobules
- The lobules are made up of acini.
- Between the acini islets of Langerhans are present. Islets of Longerhans from the endocrine part of the pancreas.
- The islets of Langerhans have alpha cells, beta cells and gamma cells.
- Endocrine secretion directly enter the blood.
- Endocrine secretions enter into the pancreatic duct system.

Islets of Langerhans :

These are cell clusters found within the pancreas. There are about 2 million islets in the human pancreas. Each islet is 200 – 300 microns in size.

Cells of islets of Langerhans :

1. Alpha cells
2. Beta Cells
3. Gamma Cells

Alpha cells form 20% of the islet cells and secretes glucogon.

Beta cells for 75% and secretes Insulin Gama cells are 1-8% and secretes gastrin.

The insulin is secreted by the beta cells. It is a polypeptide and contains 51 amino acids. Insulin maintains the blood glucose level normal. It induces glulose entry and utilization within the cell.

Pancreatic juice:

It is an alkaline fluid. About 1200-1500 ml of pancreatic juice is secreted per day.

Composition of pancreatic juice:

The pancreatic juice contains 97 to 98 % of water, trypsinogen, chymotrypsin, amylase, lipase, nuclease, carboxy peptidase.

Functions of Pancreatic juice :

- It acts on proteins, fats and carbohydrates
- Trypsinogen is converted into trypsin. Trypsin acts on proteins and convert it into proteoses and peptides. Peptones are converted into aminoacids.
- Chymotrypsinogen is converted into chymotrypsin. It acts on caesin.
- Amylase acts on starch and convert into maltose.
- Lipase acts on fats in the presence of bile salts. Bile salts convert fat into fatly acids and glycerol.
- Large amount of sodium bicarbonate is present in the pancreatic juice. This sodium bicarbonate reacts with gastric HCL.

PHYSIOLOGY

Endocrine Function of Pancreas :

The endocrine function of pancreas is performed by the islets of langerhans. Human pancreas contains about 1 to 2 million islets.

Islets of langerhans consist of four types of cells :

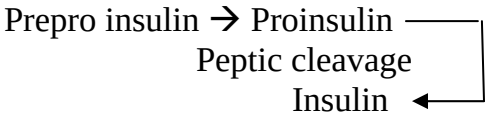
1. A or α cells which secrete glucagon
2. B or β cells which secrete insulin

3. D or δ cells which secrete somatostatin
4. F or PP cells which secret polypeptide

INSULIN

Synthesis :

Synthesis of insulin occurs in rough endoplasmic reticulum of β cells in islets of Langerhans.



ACTIONS OF INSULIN

Insulin is the important hormone that is concerned with regulation of carbohydrate metabolism and blood sugar level. It is also concerned with metabolism of proteins and fats.

1. On carbohydrate Metabolism :

Insulin is the only antidiabetic hormone secreted in the body i.e. it is the only hormone in the body that reduces blood sugar level.

The actions of insulin on carbohydrate metabolism are:

i. Facilitating transport and uptake of glucose by the cells.
ii. Increasing peripheral utilization of glucose.
iii. Increasing conversion of glucose into glycogen in liver and muscle.
iv. Inhibiting glycogenolysis.
v. Inhibiting gluconeogenesis.

2. On Protein Metabolism :

Insulin facilitates the synthesis and storage of proteins and inhibits the cellular utilization of proteins.

On Protein metabolism, Insulin :

i. Facilitates the transport of amino acid into the cell from blood – Insulin actually increases the permeability of cell membrane for amino acids.

ii. Accelerates the synthesis of proteins by influencing the transcription of DNA and by increasing the translation of mRNA.

iii. Prevents the catabolism of proteins by decreasing the activity of cellular enzymes which act on proteins.

iv. Prevents conversion of proteins into glucose. Thus insulin is responsible for conservation and storage of proteins in the body.

3. On Fat Metabolism :

Insulin stimulates the synthesis of fat. It also increases the storage of fat in the adipose tissue.

Action of insulin on fat metabolism are

i. Synthesis of fatty acids and triglycerides :

Insulin promotes the transport of excess glucose into the cells particularly the excess glucose into the cell particularly the liver cells. This glucose is utilized for the synthesis of fatly acids and triglycerides. Insulin promotes the synthesis of lipids by activating the enzymes which convert .

a. Glucose into fatty acids

b. Fatty acids into triglycerides

ii. Transport of fatty acids into adipose tissue :

Insulin facilitates the transport of fatty acids into the adipose tissue.

iii. Storage of fat :

Insulin promotes the storage of fat in adipose tissue by inhibiting the enzymes, which degrade the triglycerides.

Summary of blood glucose regulation :

1. Liver has an important blood glucose buffer system.
2. Both insulin and glucagon functions as an important feed back control system for maintaining normal glucose concentration.
3. Decreased glucose stimulates sympathetic nervous system.
4. Growth Hormone and cortisol are secreted in response to decreased blood glucose

DIABETES MELLITUS

Diabetes mellitus is a clinical syndrome characterized by hyperglycaemia due to absolute or relative deficiency of insulin.

This can arise in many different ways – lack of insulin, whether absolute or relative, affects the metabolism of carbohydrate, protein, fat, Water and electrolytes. Death may result from acute metabolic decompensation while long – standing metabolic derangement is frequently associated with permanent and irreversible functional and structural changes in the cells of the body, those of the vascular system being particularly susceptible. These changes lead in turn to the development of well defined clinical entities so called "complications of diabetes" which most characteristically affect the eye, the kidney and the nervous system.

EPIDEMIOLOGY

Epidemiological study of whole populations has shown that the distribution of blood glucose concentration is unimodal with no clear division between normal and abnormal values. Current diagnostic criteria for diabetes have been selected on the basis of identifying. These who have a degree of hyperglycaemia which if untreated has been shown to be associated with a significantly increased risk of disability and death from vascular disease, whatever the basic cause of the hyperglycaemia. The implication of these criteria is that there is no such thing as 'milk' diabetes not requiring effective treatment.

Diabetes is world –wide in distribution and the incidence of both types of primary diabetes. Insulin Dependant Diabetes Mellitus [IDDM] and Non Insulin Dependent Diabetes [NIDDM] is rising. However the prevalence of both varies considerably in difficult parts of the world. This seasons to be due to differences in both genetic and environmental factors. The prevalence in

Britain is between 1 and 2 % but almost 5% of cases of NIDDM remain undetected. The great majority of cases seen world wide have primary diabetes and Europe and North America. The ratio of NIDDM : IDDM is approximately 7:3

AETIOLOGY

Although the precise aetiology is still uncertain in both main types of primary diabetes, environmental factors interact with a genetic susceptibility to determine which of those with the genetic predisposition actually develop the clinical syndrome and timing of its onset. However both the pattern of inheritance and the environmental factors differ in IDDM and NIDDM.

IDDM

Genetics

IDDM is a heterogenous disorder in which several factors may play a role. There are the HLA system, viral infections and auto immune processes. IDDM sends to be familial disorder and there is a 25 fold increase in the risk amongst the siblings than the general population. It inheritance is strongly related to the HLA loci on chromosome 6. It is seen that HLA B_8, B_{15}, B_6, B_2, B_{W3}, DR_3 and DR_4 are associated with a higher risk of diabetes mellitus

On the other hand, a negative association has also been noted with HLA B7. In Asian Indians and Japanese, IDDM appears to be associated more with HLA B21 and BW54 than with B8. Among identical twins only 50% show concordance for IDDM as against 100% for NIDDM.

Environmental Factors :

Viruses :

Studies in mice have shown that viruses can induce diabetes by two distinct pathogenic mechanisms. Destruction of the pancreatic beta cells by

direct cytolysis results from infection with D variant of the EMC virus, Mengo virus 2T and coxasakie B4 virus induction of an autoimmune destructive process results from infection with reo virus type I and rubella.

Diet :

Bovine serem Albumin in milk may be an initiating factor in infancy when cow's milk is taken. Various nitrosoamines, coffee, glutan are also said to be factors for development of IDDM.

Auto immune mechanism :

Diabetes is seen to be associated with various autoimmune disease.

Such as Myxoedema and Addision's disease. Pancreatic islet cell antibodies have been found with multiendocrine auto immune disease.

NIDDM

Genetics :

Genetic factors play a major role in this condition. Though the exact pattern is not known, it has been variously described as autosomal dominant, autosomal recessive or polygenic. A genetic predisposition running through families is evident. Identical twins invariably develop NIDDM when exposed to the same environmental factor.

Environmental Factors:

Lifestyle :

Epidemiological studies of NIDDM provide evidence that over – eating especially when combined with obesity and under activity, is associated with the development of NIDDM. Other more direct studies have showns that middle aged diabetic patients eat significantly more and are fatter and less active than their non – diabetic siblings.

The majority of middle – aged diabetic patients are obese but only a few obese people develop diabetes. Obesity probably acts as a diabetogenic factor in those genetically predisposed to develop NIDDM.

Age :

In Britain over 70% of all cases of diabetes occur after the age of 50 years. In contrast to IDDM which mainly affects younger people, NIDDM is principally a disease of the middle aged and elderly. Thus ageing is an important risk factor for NIDDM.

Pregnancy :

The term "gestational diabetes" refers to hyperglycemia occurring for the first time during pregnancy. This may or may not disappear following delivery. Repeated pregnancy may increase the likelihood developing permanent diabetes, particularly in obese women. Long term studies show that some 80% of women with gestational diabetes ultimately development permanent clinical diabetes requiring treatment.

Sex :

Both sexes suffer equally but in lower age groups males and in middle age groups females are more affected.

Stress and Strain :

Physical and mental stress or strain may be responsible at least in precipitating the latest form of the disease as counter regulatory hormones are secreted in excess.

Pathogenesis of Diabetes Mellitus :

It is a complex affair and is still not clear. Lack of insulin, presence of insulin antagonists or excessive neoglucogenesis are possibly responsible for the development of diabetes. Due to lack of insulin, blood sugar level steadily rises and when it crosses the renal threshold level of 180 mg / 100 cc glycosuria results. Renal threshold level, however varies with age and pregnancy. Glucose increases the osmolarity of glomerular filtrate and also takes back the obligatory volume of water during elimination.

This results in profuse diuresis even upto the extent of 10 15 litres per day associated with hyponatraemia, hypokalaemia and hypomagnesaemia. Thus intense thirst, dehydration, shock and crystalloid imbalance may develop.

Again as sugar is not burnt, fat is mobilized from the adipose tissues and large quantities of free fatly acid circulate in the blood. Normally these are burnt in the liver into carbon – di-oxide and water. But as these are produced in large quantities acetyl co-enzyme-A accumulates and after condensation forms hydroxybutyric acid and acetone which accumulate in the blood and ultimately appear in urine and breath. Several hormones particularly the growth hormone may help in this process – Incompletely metabolized carbohydrate eg., pyruvic acid and lactic acid also accumulate in the blood. This condition is called diabetic ketoacidosis which often leads to coma and death.

Liver is enlarged due to fat infiltration and blood contains enormous amount of neutral fat. It has been pointed out earlier in the aetiology that various hormones may act as insulin antagonists. Apart from these, insulin and bodies may be produced in the blood which may neutralize the circulating insulin produced from the beta cells of pancreas. Lastly due to continuous loss of sugar in the urine the process of neoglucogenesis from protein may be stimulated which may result in wasting of muscles and increased urinary loss of nitrogen All these factors in different combinations may be responsible for the pathphysiology of the diabetes.

CLINICAL FEATURES :

Comparative clinical features of IDDM and NIDDM :

	IDDM	**NIDDM**
Age at onset	< 40 years	> 50 years
Duration of symptoms	Weeks	Month - Years
Body Weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Auto antibodies	Yes	No
Diabetic complications	No	10 – 20 %
Family history of diabetes	No	Yes
Other autoimmune disease	Yes	No

While the distinction between IDDM and NIDDM is broadly true in relation to the features listed, overlap occurs particularly in age at onset of diabetes, duration of symptoms and family history. Thus some young people have a form of NIDDM designated Maturity Onset diabetes in the Young while some middle – aged and elderly patients present with typical autoimmune type I IDDM.

Patients with IDDM usually show no physical signs attributable to diabetes. In the fulminating case the most striking features are those of salt and water depletion, that is loose dry skin, a furred tongue and cracked lips, tachycardia, hypotension and reduced intra ocular pressure. Breathing may be deep and sighing due to acidosis, the breath is usually fetid and the sickly sweet smell of acetone may be present. Mental apathy, confusion or coma may also be present.

The physical signs present in patients with NIDDM at diagnosis depend on the mode of presentation. Proritus vulve or balanitis is a common presenting symptom since the external genitalia are especially prone to infection by fungi which flourish on skin and mucous membranes contaminated by glucose. Ophthamoscopy may show the typical appearances of diabetic retinopathy. Depression or loss of the tendon reflexes at the ankles and impaired perception of vibration sensation distally in the legs indicate neuropathy.

Other abnormalities of neurological examination are less common. Hypertension and signs of atheroselerosis are common and may include diminished or impalpable pulses in the feet, bruits over the carotid or femoral arteries and gangrene of the feet. Signs of water and salt depletion with associated mental changes may be seen in cases with severe hypergycaemia.

Apart from patients with established clinical diabetes two other categories are recognized : Potential and latent diabetes.

Potential diabetes are persons with a normal glucose tolerance test who have an increased risk of developing diabetes for genetic reasons, e.g. an individual who has a first degree relative with diabetes.

Latent diabetics are persons in whom the glucose tolerance test is normal but who are known to have given an abnormal result under conditions imposing a burden on the islet beta cells, e.g., during pregnancy, infection or other severe stress, mental or physical during treatment with corticosteriods, thiazide diuretics or other diabetogenic drugs or when overweight.

Potential and latent diabetic patients usually complain of no symptoms and show no abnormality on examination. However certain features are recognized as being characteristic of such states without necessarily implying that such individuals will progress to clinical diabetes. For example they are predisposed to coronary and peripheral arterial disease, may show abnormal lipid patterns in response to oral contraceptives and have an increased

incidence of stillborn, abnormally large and heavy babies and babies with congenital defects.

DIAGNOSIS

Patient complains of symptoms suggesting diabetes.

- Test urine for glucose and ketones
- Measure random or fasting blood glucose.

Diagnosis confirmed by

Fasting plasma glucose > 7 mmol/1

Random plasma glucose > 11 mmol / 1

Indications for oral glucose tolerance test :

- Random plasma glucose 7 – 11 mmol / 1

INVESTIGATIONS

Examination of Urine :

1. Colour and Appearance

   Normal Urine is straw yellow in colour

2. Reaction :

   a. Litmus paper is used

The colour change

If Red - Acidic

Orange – Neutral

Yellow – Alkeline

3. Odour: Aromatic due to presence of volatile fatty acids
4. Specific gravity by urinometer – 1.010 – Increase in Glycosuria

TEST FOR GLUCOSE

Normal Valves - 24 hours specimen

- 100 mgm / 24 hours

Benedict's Qualitative Glucose test the following investigations are helpful in establishing the diagnosis of diabetes mellitus.

1. Urine Testing :

Urine tests are cheap and convenient but the diagnosis of diabetes cannot be base on urine testing alone. Urine is tested for the presence of glucose and ketones.

a. Glucosuria

Benedict's qualitative test detects any reducing substance in the urine and is not specific for glucose. More sensitive and glucose specific test is dipstick method based on enzyme coated paper strip, which turns purple when dipped in urine containing glucose.

i. Renal Glucosuria :

After diabetes the next most common cause of glucosuria is the impaired renal threshold for glucose. In such cases the blood glucose level is below 180 mg/dl, but glucose appears regularly and consistently in the urine. Renal glucosuria is a benign condition unrelated to diabetes and runs in families and may occur temporarily in pregnancy without symptoms of diabetes.

ii. Alimentary [Lag Storage] glycouria :

A rapid and transitory rise in blood glucose level above the normal renal threshold may occur in some individuals after a meal. During this period glucosuria is present. This type of response to meal is called "lag storage curve" or more appropriately alimentary glucosuria

A characteristic feature is that usually high blood glucose level returns to normal 2 hours after meal.

b. Ketonuria :

Rothera's test [Nitro prusside reaction] and strip test are conveniently performed by detection of ketonuria.

2. Single blood sugar estimation :

A grossly elevated single determination of plasma glucose may be sufficient to make the diagnosis of diabetes. Commercially available Auto – Pack kit for glucose estimation by GOD / POD Method.

3. Oral Glucose Tolerance Test :

The patient who is scheduled for oral GTT is instructed to eat high carbohydrate diet for at least 3 days prior to the test and come after an overnight fast on the day of the test first drawn. Then 75 gm of glucose dissolved in 300 ml of water is given. Blood and urine specimen are collected at half – hourly intervals for at least 2 hours. Blood or plasma glucose content is measured and urine is tested for glucosuria to determine the approximate renal threshold for glucose.

4. Intravenous GTT :

This test is performed in persons who have intestinal mal – absorption or in post – gastrectomy cases.

5. Glycosylated haemoglobin [HbA1C] :

Measurement of blood glucose level in diabetes suffers from variation due to dietary intake of the previous day. Long – term objective assessment of degree of diabetic control is better done by measurement of glycosylated hemoglobin [HbA1C] a minor hemoglobin component present in normal persons. This is because the non enzymatic glycosylation of hemoglobin takes place over 120 days, life span of red blood cells. HbA1c assay, there more gives an estimate of diabetic control for the last 3 months.

COMPLICATIONS OF DIABETES :

- **Vascular Complications :**

These are very common, cerebral, coronary, renal, limb arteries and abdominal aorta may be affected by the process of arteriosclerosis. Due to this, there may be ischaemic heart disease with or without thrombosis, cerebrovascular accident [CVA] ischaemic limb with or without gangrene, renal astery stenosis etc.

- Diabetic Ketoacidosis

It is rare now – a – days and is seen only in neglected IDDM cases precipitating factors

Stressful conditions, acute infections, myocardial infraction, trauma, neglet in the treatment, heavy carbohydrate meals etc.,

Clinical Features :

Patient is usually dehydrated, with sunken eyes, dry skin and tongue and prominent malar bones. The ocular tension is low – [krauss's sign]. There is kussmaul's air hunger with hissing respiration. BP is usually low, pulse is low in volume. Abdomen may be rigid and tender. Smell of acetone is present in the breath.

Evidence of infection e.g., boils, carbuncles and respiratory tract infection are very common. Patient may be conscious initially but gradually drowsiness and coma supervene.

Diabetic retinopathy :

Diabetic retinopathy is the most common cause of blindness in adults between 30 and 65 years of age in developed countries.

Clinical Features :

- Micro aneurysms
- Retinal haemorrhage
- Hard Exudates
- Soft exudates
- Venous changes
- Neo vascularisation
- Pre – retinal haemorrhage
- Vitreous hemorrhage
- Fibrosis

Micro aneurysms :

They appear as minute, discrete, circular, dark red spots near to but apparently separate from the retinal vessels. Minute aneurysms arising from the venous end of capillaries

Haemorrhage :

Occur in the deeper layers of the retina and hence are round and regular in shape and described as “blot” hemorrhage.

Hard exudates :

They vary in size from tiny specks to large confluent patches and tend to occur particularly in the perimacular area they results from leakage of plasma from abnormal retinal capillaries.

Soft exudates

Sometimes referred as “cotton wool spots” these are similar to those seen in hypertension and also occur particularly within five disc diameters of the optic disc.

Neo vascularisation :

This may arise from mature vessels on the optic disc or the retina in response to areas of ischaemic retina.

Venous change :

These include dilation "beading" and increased tortuosity including "oxbow lakes" or loops. These latter changes often indicate widespread capillary non-perfusion.

DIABETIC NEUROPATHY

Central nervous system is affected in long term diabetes the clinical impact of diabetes is mainly manifest on the peripheral nervous system.

Pathology :

- Axonal degeneration of both myelinated and unmyelinated fibers.
  - Early : axon shrinkage
  - Late : axonal fragmentation, regeneration
- Thickening of schwann cell basal lamina.
- Patchy, segmental demyelination
- Abnormalities of intraneural capillaries :
  Thickening of basement membrane and micro thrombi.

Classification of Diabetic neuropathy :

Somatic :

1. Poly Neuropathy

i. Symmetrical mainly sensory and distal

ii. Asymmetrical mainly motor and proximal.

2. Mono Neuropathy.

Visceral [Autonomic]

i. Cardio Vascular

ii. Gastro intestinal

iii. Genitourinary

iv. Sudomotor

v. Vaso motor

vi. Pupillary

Clinical Features

Symmetrical sensory Polyneuropathy :

The most common signs found on physical examination are loss of tendon reflexes in the lower limbs, diminished perception of vibration sensation distally and "glove and stocking" impairment of all other modalities of sensation. Symptoms include paraesthesiae in the feet and sometimes in the hands, pain in the lower limbs, burning sensations in the sole of the feet, cutaneous hyper aesthesia and abnormal gait often associated with a sense of numbness in the feet.

Asymmetrical motor diabetic neuropathy :

This presents as severe and progressive weakness and wasting of the proximal muscles of the lower limbs. Hyper aesthesia and paraethesiac are also common.

Mono Neuropathy :

The nerves most commonly affected are : The third and sixth cranial nerves resulting in diplopia due to impaired ocular movement. The ulnar and median nerves leading to the clinical picture of carpal tunnel syndrome.

Autonomic neuropathy :

Parasympathetic nerves or sympathetic nerves may be predominantly affectly in any one or more system.

Lactic Acidosis :

It is characterized by accumulation of excess of lactic acid in the blood. Normally lactic acid is derived from RBC, skeletal muscles, skin and brain. Removal of lactic acid occurs through kidneys and liver. Excessive production of lactic acid as occurs in tissue hypoxia, defective removal as occurs in hepatic failure or in circulatory collapse both may cause excessive lactic acid accumulation in blood. In conditions like Hepatic failure, Renal failure, CCF, Respiratory failure, septicemia, Infarction of long segment of intestine or limb may produce lactic acidosis.

Clinical features :

On set is rapid. Symptoms include hyper ventilation and vascular collapse. In idiopathic spontaneous variety however BP is normal and vascular collapse does not develop.

Renal Complications :

i. Pylo nephritis :

This is very common and may lead to chronic renal failure afterwards.

ii. Renal arteriosclerosis

iii. Papillitis Necroticans :

The renal papillae will show necrosis, ultimately leading to uraemia.

iv. Kimmelstiel Wilson Syndrome : [K.W. Syndrome]

This is an important complication which develops usually in long standing cases. Clinically patient will present features of nephnotic syndrome.

v. Micro albuminuria

vi. Glycogen in renal tubule, fibrin cap and capsular drop are other renal complications which do not produce any specific clinical feature but can be diagnosed by renal biopsy.

Sexual and Genital Complications :

Impotency and frigidity may develop. Balanitis and Balanoposthitis are also very common complications in males. These are due to secondary infection as urine contains heavy amount of sugar and nitrogenous materials Leucorrhoea may develop in females.

Pulmonary Complications :

Tuberculosis is very common in diabetes in our country and this may remain asymptomatic. Skia gram of the chest is a must in all cases of diabetes mellitus. Other infective complications like pneumonia bronchopneumonia, supportive pneumonitis, pleurisy etc., may also develop.

Effects on pregnancy and Neonates :

There may be miscarriages and abortions, toxemias of pregnancy, hydraminos etc., Herculin child may be born of diabetic mothers due to secretion of excess of growth hormone and maternal hyperglycaemia. There is increased perinatal mortality, increased incidence of respiratory distress syndrome and infection. Neonates may have a characteristic attitude also.

Diabetic cataract :

There may be senile cataract with clouding of the lens or radially arranged cart – wheel opacities. Sometimes true diabetic cataract may develop which is snow – flake in appearance and is seen in juvenile cases. This does not require surgery for treatment.

Skin Complications :

Skin infections are very common as sugar in tissues and sweat act as a good media for bacterial growth. Boils, carbuncles and fungal infections particularly monilial infection of vulva are very common. Hypercholesterolaemia may give rise to

Xanthoma Diabeticum with nodule :

Xanthosis Necrobiosis lipoidica diabeticorum may also develop but rare. Shin spots may be seen in adult diabetics.

Non – Ketotic hyperosmolar diabetic coma :

This is a serious emergency seen occasionally in elderly diabetics. The condition is precipitated by infections, myocardial infarction, burns, trauma, surgical stress, renal failure, pancreatitis and the use of certain drugs like thiamine diuretics, steroids and propranolol. In this condition probably a small amount of endogenous insulin is present so that lipolysis does not occur, but glucose metabolism is grossly deranged. Blood sugar level often exceeds 600 mg / dl.

Keto acidosis is absent and this distinguishes the condition from diabetic coma, plasma osmolarity rises above 360 mos mol / liter and there is profound cellular dehydration. The haematocrit is high.

When the fluid loss becomes severe, hypernatremia develops. Body potassium is lost and a total deficit of 40 to 100 mEq may occur.

The normal plasma osmolarity ranges from 280 to 310 mos mol / L. Valves above 340 mos mol/L are abnormal. In severe cases the osmolarity may exceed 370 mos mol. This condition starts with extreme weakness and drowsiness the patient slips into coma. Other neurological manifestations such as seizures, ataxia, hemiparesis, aphasia and mental disturbances may be present. Though thirst is present in early stages.

MANAGEMENT :

There are three options for the mode of therapy to be initiated.

1. Diet and exercise
2. Oral hypoglycemic agents
3. Insulin

Nutritional approach :

General consensus on proportion of food constituents is as follows :

- Carbohydrates 60 – 65%
- Fat 20 – 25%
- Protein 10 – 15%
- Excessive salt intake > 6g / day should be discouraged especially in those with hypertension.
- Protein intake should be o.8 g/kg for those with nephropathy.
- Non – nutritive sweetness eg: saccharin, aspartam can be used in moderation.
- Alcohol intake should be in accordance with general health preview, on social occasions and in moderation. In pregnancy, those susceptible to hypoglycemia and those on chlorpropamide should avoid alcohol.

Those being initially advised diet and exercise programme needs considerable inputs by physician, nutrition counsellor and physical exercise experts. If there is motivation and patient empowerment achieved, results for weight reduction and normalization of blood glucose are remarkable.

Diet plan should be flexible, reflection patient's life style, work schedule and local meal preparation facilities.

Calorie intake should be appropriate to desirable body weight. The diet should be balanced with plenty of natural availability of minerals and vitamins.

It should have a variety and be pleasant to taste. "Special Diabetes" food products are unnecessary.

Exercise :

Regular physical exercise has important physiological and psychological benefits for all diabetes.

The extent of frequency and severity of exercise need elaboration. It should be followed 5 days a week for duration of 40 minutes each day and its severity should achieve 50 – 70% of individuals maximum of uptake.

While children should take part in team games, adults may undertake individually suitable form : jogging, swimming, golfing, cycling, yoga etc. Those exercising should be aware of the risk of hypoglycemia, cardiac arrhythmia or lower extremity injuries.

Those with existing vascular complication may seek physician's guidance as to the schedule for exercise.

Exercise has following beneficial effects :

1. It improves muscle tone, increases work capacity and reduces fatigue.
2. There is increase in cardio – respiratory reserve.
3. Insulin sensivity improves, anti diabetic medication can be reduced.
4. There is reduction in LDL and increase in HDLC with exercise.
5. Exercise provides a sense of well being and gives psychological back– up.

MATERIALS AND METHODS

The study was carried out in "Post Graduate Department of Maruthuvam", Government Siddha Medical College S.Arignar Anna Hospital of Indian Medicine" Chennai 600 106.

During this dissertation work on Madhumegam [Diabetes Mellitus] twenty patients were examined and treated as In patients in the hospital and twenty others in the out Patient Department. They were with clinical symptoms and signs of Madhumegam, comprising of both sexes above thirty years of age. The study was conducted for a period of six weeks under the guidance of the professor and the Reader of "Post Graduate Department of Maruthuvam".

CRITERIA FOR SELECTION:

- Above 30 years of age
- Both sexes
- Non Insulin Dependent Diabetes Mellitus
- Polyuria
- Polyphagia
- Polydipsia
- Nocturia
- General weight loss
- General weakness

CRITERIA FOR EXCLUSION:

- Insulin – Dependent Diabetes Mellitus
- Patients with hyperglycemia due to Secondary, causes like pancreatic pathology, patients with cardiovascular diseases
- Tuberculosis

- Diabetic Nephropathy
- Diabetic Retinopathy

SIDDHA SYSTEM OF CLINICAL DIAGNOSIS:

- Poriyal Therthal – Mei, Vai, Kann, Mooku, Sevi
- Pulanal Therthal – Unarthel, Suvaithal, Parthal, Mugarthal, Kettal
- Vinnathal
- Mukkutra Nilaigal – Vali, Azhal, Iyam
- Ezhu Udal Kattukal – Saaram, Senner, Oon, Kozhuppu, enbu, Moolai, Sukkilam
- Envagai Thervu – Naa, Niram, Mozhi, Sparism, Malam, Moothiram, Naadi.

CASE SHEET PROFORMA:

Twenty patients were treated as In-patient with clinical signs and symptoms of Madumegam.

- Complaints and Duration
- History of present illness
- Personal habits
- Personal History
- Family history
- Systemic examination
- Laboratory Investigation
- Prognosis of the disease and Management

INVESTIGATION:

The next step in Research Oriental Programme is Investigation, to confirm the diagnosis predicted. The Investigations were carried out promptly and regularly before and after treatment.

All patients were subjected to routine clinical investigation which include:

- Urine Sugar – Fasting and post prandial.
- Albumin and Deposits in Urine and
- Ova and Cyst in Motion
- Total count, Differential count, Erythrocyte sedimentation rate, Haemoglybin and urea, cholestrol in blood
- Blood sugar, fasting and post – prandial by GOD – POD Method other investigations like glucose tolerance test, Glycosylated Haemoglobin, X– ray chest, ECG and Eye – Fundus are also carried out if necessary.

TEST FOR GLUCOSE IN URINE:

Normal Valves - 24 Hours Specimen

\- 100 mg / 24 Hours

Benedict's Qualitative Glucose test:

In this method, the cupric ion is reduced to cuprous oxide [CU_2O]. If only 0.1% of less of glucose is present the precipitate may not appear until cooling.

To 5ml of Benedict's qualitative reagent add 8 drops of urine [0.5ml] and boil it over a flame for 2 minutes. Blue to cloudy green colour– Negative

Yellow Green	+	[<0.5% Glucose]
Greenish Yellow	++	[0.5% - 1% Glucose]
Yellow	+++	[1.2% Glucose]
Orange to Brick Red	++++	[over 2% glucose]

BLOOD SUGAR ESTIMATION:

Commercially available Auto – Pak kit for glucose estimation by GOD / POD Method.

Intended Use:

The reagent kit is intended for in – vitro quantitative determination of glucose in serum or plasma.

Principle :

Glucose is oxidized by glucose oxidase [GOD] into gluconic acid and hydrogen peroxide. Hydrogen peroxide in presence of peroxide [POD] oxidizes the chromogen 4 – amino – phenazone / phenolic compound to a red coloured compound. The intensity of the red colour produced is proportional to the glucose concentration and is measured at 505 nm [40 / 90 – 530 nm]

The final colour is stable for 2 hours

GOD

Glucose + O_2 -----→ Glucose Acid + H_2O_2

H_2O_2 + Phenolic compound +4 – amino

POD

- Phenozone ------→ Red Compound $2H_2O$

Sample Collection:

Fresh serum or plasma is preferred for glucose determination.

Reagents:

Tablets:

Buffer / Enzymes [GOD / POD]

Chromogen [4 – aminophenozone, phenolic compound]

Standard : Glucose 100 mg / dl ready for use preparation of working solution for 20ml tablet.

Gently dissolve 1 tablet in 20 ml of distilled water in a clean beaker, with continuous stirring. Transfer the solution into a dark bottle and label this solution as working solution.

Procedure :

The samples and the working solution should be brought to room temperature prior to use. 1 ml of the working solution is mixed with .01 ml of serum in a test tube and the readings are taken from semi – auto analyzer. [Slim seal].

Expected Valves :

Serum / Plasma 70 100 mg / dl [Fasting]

All these data were recorded systematically in the case sheet proforma for analysis.

DRUG AND DOSE SCHEDULE

Sarabanthra Madhumega Choornam	-	1 gm with hot Water – Thrice daily after food.
Madhumega Kudineer	-	30 ml twice daily before food.

The Biochemical constituents and microbiological effects of the drug were studied. The drug was also subjected to the pharmacological and toxicological tests in rat models

PREPARATION OF TRIAL MEDICINE

I. SARABENDRA MADHUMEGA CHOORNAM – 1gm tds with hot water - After meals

II. MADHUMEGA KUDINEER – 30 ml BD –Before meals

PREPARATION OF SARABENDRA MADHUMEGA CHOORNAM:
Ref: Sarabendra Vaidya rathnavali

Drugs:

S.No	Name	Botanical Name	Part Used	Quantity
1.	Murungaipattai	Moringa Pterygosperma	Bark	70gms
2.	Karunesembaipattai	Sesbania Aegyptiaca	Bark	70gms
3.	Kothimai Sathu	Triticum Vulgane	Seed	140gms
4.	Karuvelampisin	Acacia Arabica	Pisin	70gms
5.	Maruthani Vithai	Lawsonia alba	Seed	70gms
6.	Manjal	Curcuma longa	Underground steam	1.4 kg
7.	Salamisiri	Orchis Masoula	Dried root of the orchis masoula	1.4kg

The above drugs are dried and powdered and mixed well and sieved using a cloth – Vastrakayam

Dose- 1gm tds with hot water after meals

Life span of Choornam : 3 months

MADHUMEGA KUDINEER
Ref Mooligai marmam – III part

Drugs:

S.No	Name	Botanical Name	Part Used	Quantity
1.	Avarampattai	Cassia auticulata	Bark	500gms
2.	Aathipattai	Ficus glomavata	Bark	500gms
3.	Maruthampattai	Terminalia arjuna	Bark	500gms
4.	Sarakondaraipattai	Cassia fistula	Bark	500gms

The above raw materials were coarsely powdered uniformly mixed and stored in airtight container.

For every dose the decoction was made using 15gm of the above powder with 120ml of water and reduced to 30ml by boiling, Filtered and administered to each patient before meals twice a day.

Dose: 30ml B.D before Food

Life span of kudineer – 3hours

INDIVIDUAL DRUG STUDY

1. fUnty« ÃÁ‹ (Gum of Acacia Arabica)

Rit	:	Jt®¥ò
j‹ik	:	j£g«
ÃçÎ	:	Ïå¥ò
brŒif	:	cŸsHyh‰¿, tu£Áaf‰¿, clYukh¡», Eiupuš, nehaf‰¿, M©ik bgU¡».
Fz«	:	ÏJ Ú®¤J¥nghd jts¤ij ÏWf¢ brŒÍ«. vç¢rnyhL éG»‹w Óœ btŸisia ãW¤J«.

“Ú¤bjhGF« éªj ãiy¡f¥ òçÍbkç

ó¤bjhGF« btŸisjid¥ ngh¡Fäl« kh¤Âunkh

njR jUKuŠ brŒÍ« bgçnahuh‰

ngRfU nty« ÃÁ‹” - mf¤Âa® Fzthfl«

2. **kŠrŸ (Curcuma longa)**

Rit	:	fh®¥ò, if¥ò
j‹ik	:	bt¥g«
ÃçÎ	:	fh®¥ò
brŒif	:	mf£LthŒ mf‰¿, bt¥gK©lh¡»
Fz«	:	Ïjdhš thªÂ, të, Ô, Ia¡F‰w«, jiytè, Únu‰w«, btŸis, _¡FÚ® ghŒjš, Itif tè, Å¡f«, t©L¡fo, bgU«ò© Ïit ngh¡F«.

“bgh‹åwkh« nkå òyhdh‰w K«nghF«

k‹D òUl tÁakh« - Ã‹åbaG«

thªÂÃ¤j njhlika« thj« nghª Ôgdkh§

T®ªjkŠr ë‹»H§F¡ F.

-mf¤Âa® Fzthfl«.

3. **rhyhäÁç (Orchis masoula)**

Rit	:	Ïå¥ò

j‹ik : j£g«

ÃçÎ : Ïå¥ò

brŒif : fhk¥ bgU¡», clš cukh¡»

Fz« : ÏJ R¡»y éU¤ÂiaÍ«, clš t©ikiaÍ« c©lh¡F«.

4. **kUjhâ éij : (Lawsonia alba)**

Rit : Jt®¥ò

j‹ik : bt¥¥g«

ÃçÎ : fh®¥ò

brŒif : eh‰wkf‰¿, óÂfªj ehrå

Fz« : ÃšèNåa«, ngŒ, ój§fŸ Ït‰¿‹ brŒif bfL«.

“kUnjh‹¿ æ‹éij¡F khÃšiy N‹a«
éUjh©l ngŒój« nkÎ«”

- mf¤Âa® Fzthfl«.

5. **nfhJik r¤J (Triticum Vulgare)**

Rit : Ïå¥ò

j‹ik : j£g«

ÃçÎ : Ïå¥ò

brŒif : cŸsHyh‰¿

Fz« :

“nfhJikæ‹ e‰Fzªjh‹ nfhÂ‰ gy§bfhL¡F«
jhJé®¤Â ah¡Fª jåthŒit¢ nrÂ¡F«
Ã¤j« më¡F« Ãunkf¤ ijbfL¡F«
c¤jkh« v‹nw ciu” - mf¤Âa® Fzthfl«

6. **KU§if¥g£il (Bark of Moring Pterygosperma)**

Rit : if¥ò, Jt®¥ò, Ïå¥ò

j‹ik : j£g«

ÃçÎ : fh®¥ò

brŒif : fU¥g¢ Áij¢Á

Fz« : të F‰w«, Áy eŠRfS« ÔU«.

“KU§ifnt®¥ g£il¡F _L fg¤njh
blhU§F»w¢ rªãfu« XL« - mU§fdf
t£il¥ bghUKiyahŒ thŒbthLél§fS nk‰
g£il¡F¥ nghnk gwªJ”

- mf¤Âa® Fzthfl«

7. **fUŠbr«ig¥g£il (Bark of Sesbania Aegyptiaca)**

Rit : if¥ò, Jt®¥ò

j‹ik : bt¥g«

ÃçÎ : fh®¥ò

brŒif : bt¥g K©lh¡», UJÎ©lh¡», Jt®¥Ã, òG¡bfhšè, ÁWÚ®¥bgU¡», Å¡f§ fiu¢Á

Fz« :

“é¥òUÂ¥ ò©zhW« ÅDfu¥ ghD«nghª
j¥ghkš nkfª jâÍ§fh© - bt¥gh®
fgnuhf nkUŠ fUŠbr«ig bah‹W¡
»gkh Kiykhnj v©”

8. **Mthu«g£il (Bark of Cassia auriculata)**

Rit : Jt®¥ò

j‹ik : j£g«

ÃçÎ : Ïå¥ò

brŒif : Jt®¥ò, cukh¡»

Fz« : Ïij fõhaä£L thŒ bfh¥gë¡fÎ«, MrdtêahŒ Õ¢rÎ« cgnah»¡fÎ«.

9. **m¤Â¥g£il (Bark of Ficus Glomvata)**

Rit : Jt®¥ò

j‹ik : j£g«

ÃçÎ : Ïå¥ò

brŒif : Jt®¥Ã

Fz« : ÑœthŒ¡fL¥ò, FUÂ¥ngh¡F, Ój¡fê¢rš, eh‰wKŸs ò©fŸ, btŸis M»aitfis ngh¡F«.

“ÅW fL¥Ãu¤j« bt©Ój u¤jbkhL
ehWéu z§fbsšyh« ehlhth« - TW§fhš
m¤ÂjU nkf«ngh« MæiHna! vŠPh‹W«
m¤Â¥gh‰ g£il¡ f¿”

- mf¤Âa® Fzthfl«

10. **kUj«g£il (Bark of Texminalia arjuna)**

Rit	:	Jt®¥ò
j‹ik	:	j£g«
ÃçÎ	:	fh®¥ò
brŒif	:	cukh¡», jkuf bt¥gK©lh¡»
Fz«	:	ÚçêÎ, btŸis, ka¡f«, Ú®nt£if, Ru¤ÂY©lh¡F« ja¡f«, bgU nehŒ, òGnehŒfŸ, tæ‰W nehŒ, tw£Niy Ïit ngh« jkuf¤ij t‹ik¥ gL¤J«.

“XjbkD Úêêit nah£L« Ãunkf§
fhjbkd nthl¡ fl¤J§fh©- nghj
ka¡f bkhLjhf khwh¢ Ru¤Â‹
ja¡fkWf F« kUjŠ rh‰W”

- mf¤Âa® Fzthfl«

11. **ru¡bfh‹iw¥g£il (Bark of Cassia Fistula)**

Rit	:	if¥ò, Jt®¥ò
j‹ik	:	bt¥g«
ÃçÎ	:	fh®¥ò
brŒif	:	kyäs¡», Jt®¥Ã, vç¢rš c©lh¡»
Fz«	:	bgUnehŒ, òG, Niy, K¡F‰w«, brçahik, M»ait ngh« - ÏJ fêa¢ brŒÍ«.

“F£l§ »Uä bfhLŠNiy thjika«
J£l kykUÂ öu¥ngh« - j£oŠ
Ru¡»‹w ngÂÍ©lh« JŒ¡f¤ Jt®¡F«

Ru¡bfh‹iw¡ fhuz§nf! rh‰W

- mf¤Âa® Fzthfl«

CHEMICAL ANALYSIS OF HERBAL PREPARATION

Preparation of Extract

5gm of Sarabenthra Madhumega Choornam and Madhumega Kudineer is weighed accurately and placed in a 250ml clean beaker and added with 50ml of distilled water. Then it is boiled well for about 10 minutes. Then it is cooled and filtered in a 100ml Volumetric flask and made up to 100ml with distilled water.

S.No.	Experiment	Observation	Inference
I.	**Test for Acid Radicals**	Absence of white precipitate	Absence of sulphate
1.	Test for Sulphate		
a.	2 ml of the above prepared extract is taken in a test tube. To this add 2		

	ml of 4% Ammonium oxalate solution.		
b.	2 ml of sodium carbonate extract as added with 2 ml of dilute Hydrochloric acid is until the effervescence ceases off. Then 2 ml of Barium chloride solution is added.	Absence of cloudy appearance in concentrated	Absence of Sulphate
2	**Test for Chloride:** 2ml of Sodium carbonate extract is added with dilute Nitric acid till the effervescence ceases. Then 2 ml of silver Nitrate solution is added.	Absence of cloudy white precipitate	Absence of Chloride
3.	**Test for Phosphate:** 2 ml of the extract is treated with 2 ml of Ammonidum Molybate solution and 2ml of concentrated Nitric acid.	Absence of Yellow precipitate	Absence of Phosphate
4.	**Test for carbonates:** 2 ml of the extract is treated with 2ml of Magenisum sulphate solution.	Absence of white precipitate	Absence of Carbonate
5.	**Test for Sulphide:** 1 gm of the substance is treated with 2 ml of concentrated Hydrocholric acid	Absence of rotten egg smelling gas	Absence of Sulphate

6.	**Test for Nitrate;** 1 gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of Reddish brown gas	Absence of Nitrate
7. a)	**Test for Fluoride and Oxalate:** 2 ml of the extract is added with 2 ml of dilute Acetic acid and 2 ml of calcium chloride solution and heated.	Absence of white precipitate	Absence of Fluoride
b)	5 drops of clear solution is added with 2 ml of dilute sulphuric acid and slightly warmed. To this, 1 ml of dilute Potassium permanganate solution is added.	No change in potassium permanganate solution colour	Absence of Oxalate
8.	**Test for Nitrite:** 3 drops of the extract is placed on a filter paper. On that, 2 drops of Acetic acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absence of Nitrate
9.	**Test for Borate:** 2 pinches of the substance is made into paste by using sulphuric acid and alcohol (95%) and introduced in to the blue flame.	Absence of Green tinged flame	Absence of Borate
II.	**Test for Basic Radicals**		
10.	**Test for Lead:** 2 ml of the extract is added with 2 ml of Potassium lodine solution.	Absence of Yellow precipitate	Absence of Lead
11. a.	**Test for Copper** One pinch of substance is made in to paste with concentrated Hydrochloric acid in a watch glass and introduced in to the nonluminous part of the flame.	Absence of slight bluish green flame	Absence of Copper
b.	2 ml of the extract is added with excess of Ammonia solution.	Absence of Bluish precipitate	Absence of Copper

12.	**Test for Aluminium:** To the 2 ml of extract, soldium hydroxide solution is added in drops to excess.	Absence of white precipitate	Absence of Aluminium
13. a.	**Test for Iron:** To the 2 ml of extract, 2 ml of Ammonium thiocyanate solution is added.	Presence of Blood red colour	Presence of Ferrous Iron
b.	To the 2 ml of extract 2 ml of Ammonium thiocyanate solution and 2 ml of concentrated Nitric acid added.	Presence of Blood red colour	Presence of Ferrous Iron
14.	**Test for Zinc:** To the 2 ml of extract sodium hydroxide solution in added in drops to excess.	Absence of white precipitate	Absence of Zinc
15.	**Test for calcium:** 2 ml of the extract is added with 2 ml of 4 % Ammonium Oxalate solution.	Absence of white precipitate	Absence of Calcium
16.	**Test for Magnesium:** To 2 ml of extract, sodium hydroxide solution is added in drops to excess.	Absence of white precipitate	Absence of Magnesium
17.	**Test for Ammonium:** To 2 ml of extract few ml of Nessler's reagent and excess of sodium hydroxide solution are added.	Absence of reddish brown precipitate	Absence of Ammonium
18.	**Test for Potassium:** A pinch of substance is treated with 2 ml of sodium nitrate solution and then treated with 2 ml of cobalnitrate in 30% glacial Acetic acid.	Absence of yellow precipitated	Absence of Potassium
19.	**Test for Sodium:** 2 pinches of the substance is made in to paste by using Hydrochloric acid and introduced in to the blue flame.	Absence of Yellow colour flame	Absence of Potassium

20.	**Test for Mercury:** 2 ml of the extract is treated with 2 ml of sodium hydroxide solution.	Absence of Yellow precipitate	Absence of Mercury
21.	**Test for Arsenic:** 2 ml of extract is treated with 2 ml of Silver nitrate solution.	Absence of Yellow/Brownish red precipitate	Absence of Arsenic
III.	**Miscellaneous:**		
22.	**Test for Starch:** 2 ml of extract is treated with weak Iodine solution.	Blue colour develops	Presence of Starch
23.	**Test for reducing sugar:** 5 ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 8 to 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	No Change in colour	Absence of reducing sugar
24. a.	**Test for alkaloids:** 2 ml of the extract is treated with 2 ml of Potassium iodide solution.	Presence of Red colour	Presence of Alkaloid
b.	2 ml of extract is treated with 2 ml of picric acid	Absence of Yellow colour	Absence of Alkaloid
25.	**Test for Tannic acid:** 2 ml of the extract is treated with 2 ml of Ferric chloride solution.	Presence of black precipitate	Present of tannic acid
26.	**Test for unsaturated compound:** To 2 ml of the extract 2 ml of Pottassium permanganate solution is added.	Potassium permanganate decolorised.	Presence of unsaturated compound
27.	**Test for Amino acid:** 2 drops of the extract is placed on a filter paper and dried well. After drying 1% Ninhydrine is sprayed over the same and dried well.	Absence of Violet colour	Absence of Amino acid

28.	**Test for Albumin:** 2 ml of the extract is added with 2 ml of Esboch's reagent.	Absence of yellow precipitate	Absence of Albumin
29.	**Test for Type of Compound:** 2 ml of the extract is treated with 3 ml of Ferric chloride solution.	Absence of red colour	Absence of Anti pyrine, Aliphatic Amino acid

RESULTS:

The given sample contains:

ACID RADICALS:

Absence of acid radials

BASIC RADICALS

Iron Present

MISCELLANEOUS:

Starch, Alkaloids, Tannic acid, unsaturated compound present

ACUTE TOXICITY STUDY

TOXICOLOGICAL EVALUATION FOR SARABENTHRA MADHUMEGA CHOORNAM AND MADHUMEGA KUDINEER POWDER

Acute oral toxicity study (Ecobichnon, 1997)

The procedure was followed by using OECD guidelines (Organization of Economic Cooperation and Development) 423 (Acute Toxic Class Method). The acute toxic class method is a stepwise procedure with 3 small animals of a single sex per step. Depending on the mortality and or morbidity status of the animals, on the average 2-4 steps may be necessary to allow judgement on the acute toxicity of the test substance. This procedure results in the use of a minimal number of animals while allowing for acceptable data based scientific conclusion. The method, uses, defined doses 2000 mg/kg body weight. The results allow a substance to be ranked and classified according to the Globally Harmonized System (GHS) for the classification of chemicals which acute toxicity.

Experimental procedure

Female wistar rats weighing 150 – 200 gm were used for the study. The starting dose level of Sarabenthra madhumega Choornam and madhumea kudineer Powder was 2000 mg/kg body weight per oral (P.O).As most of the crude extracts posses LD_{50} value more than 2000 mg/kg per oral(O.P) the starting dose used was 2000 mg/kg p.o. Dose volume was administered 0.1 ml/10 gm body weight to the rat which were fasted night over with water *ad libitum*. Food was withheld for a further 3-4 hours after administration and observed for signs of toxicity. Body weight of the rats before and after termination were noted aand any changes in skin and fur, eyes and mucous membrane and also respiratory, circulatory, autonomic and central nervous systems and somatomotor activity and behaviour

pattern were observed and also signs of tremors, convulsion, salivation, diarrhoea, lethargy, sleep and coma were noted. The onset of toxicity and signs of toxicity also noted.

Result

The trial drug Sarabenthra Madhumega Choornam and Madhumega Kudineer powder did not exhibit any significant toxicity at 2000 mg/kg body weight. So the drug is safe for long term administration.

ANTI DIABETIC OF MADHUMEGA CHOORNAM AND MADHUMEGA KUDINEER AGAINST STREPTOZOTOCIN INDUCED DIABETIC RATS

Experimental animals

Species	-	Wistar rats
Sex	-	Male
Weight	-	150-200g
Drug	-	Streptozotocin (Sigma)

Induction of diabetes Mellitus

After fasting for 18 hours, 30 rats were injected by intraperitoneally with a single dose of 50 mg/Kg Streptozotocin after dissolving if in freshly prepared ice-cold citrate buffer (p.H.4.5). After the injection, they had free access to feed and water and were given 5% glucose solution to drink overnight to counter the hypoglycemic shock.

The development of diabetes was confirmed after 48 hr of the streptozotocin injection. The animals having fasting blood glucose level more than 200 mg/dl were selected for the experimentation. From the out of 30 animals, 2 animals were died before grouping and one animal was omitted from the study, because of mild hyperglycemia (168 mg/dl). From the 27 diabetic animals, they were divided into 3 groups each having 9 animals.

Collection of blood sample and glucose determination

Blood samples were collected by end tail vein cutting method and blood glucose level was determined by using one touch electronic glucometer using glucose test strips. This method that permits the measurement of blood glucose levels with a minimum injury to the rat, this method was previously validated by comparison with GOD – POD Chromogen method.

Experimental Protocol:

The Group I conists of 6 normal control animals. The remaining 3 groups consist of 9 streptozotocin induced diabetic rats.

Group I : Normal control animals received 1% SCMC 5ml / kg body wt. orally for 15 days.

Group II : Streptozotocin induced diabetic animals received % SCMC 5ml/kg b.w. per orally for 15 days.

Group III : Streptozotocin induced diabetic animals received Madhumeha Churnam 180 mg/kg and Madhumega Kudineer 2.7 ml/kg b.w per orallyfor 15 days.

Group IV : Streptozotocin induced diabetic animals received glibenclamide 1.25 mg/kg b.w. per orally for 15 days.

All the group of animals received the treatment by the bove schedule for 15 days. Blood samples were collected in morning one hour after drug administration and day 5, 10 & 15 to determine the blood glucose level by using electronic glucometer.

EFFECT OF BLOOD GLUCOSE LEVEL ON TREATMENT OF MADHUMEHA CHOORNAM AND MADHUMEGA KUDINEER AGAINST STREPTOZOTOCIN INDUCED DIABETIC RATS

S.No.	Group	Blood glucose level (mg/dl)			
		Initial	5th day	10th day	15th day
1.	I (n=6)	67.85 ± 2.54	72.5 ± 4.5	74.0 ± 2.65	72.15 ± 8.82
2.	II (n=5)	247.5 ± 17.56	286.32 ± 12.35*	325.5 ± 25.80*	330 ± 21.90*
3.	III (n=6)	252.6 ± 15.15	258.5 ± 8.7^{ns}	181.7 ± 8.9*	142.0 ± 8.75*
4.	IV (n=7)	250.0 ± 12.5	215.3 ± 8.50*	156.8 ± 6.08*	130.5 ± 5.86*

The values expresses mean ± sem. n = Number of animals in each group.

Statistical signifance were carried out byAnova followed by Dunnet's Test.

*p<0.05, ns – non significant

Result:

The blood glucose level of the streptozotocin administered (Group-II) animals signicantly (P<0.05) increase when compare with normal control animals (Group I). The blood glucose level of Sarabanthra Madhumega Choornam and Madhumega Kudineer administerered animal not excitibited – P-124. Significant observation on 5th day. The blood Glucose level of Sarabenthra Madhumega Choornam and Madhumega Kudineer administered animal excihbited significant response on 10th & 15th day.

Reference:

1. Brosky G, Logothelopoulos J. Streptozotocin induced diabetes in the mouse and guinea pig. Diabetes 1969, 18, 606 – 609.
2. BabuV, Gangadevi T, Subramoniam A. Antihyperglycemic effect of *Cassia Keinii* leaf extract in glucose fed normal rats nd alloxan induced diabetic rats. Ind. J. of Pharmacol. 2002: 34:409-415.
3. Rajesh Kumar G., Achyut Narayan K., Geeta W., Murthy P.S.Ramesh C., Kapil M and Vibha T. Hypoglecemic and Antidiabetic effect of a queous extract of leaves of *Annona Sqamosa* (L) in experimental animals. Current science, 2005; 88, No.8.

POST – GRADUATE DEPARTMENT BRANCH I MARUTHUVAM
GOVERNMENT SIDDHA MEDICAL COLLEGE
CHENNAI – 106
CASE SHEET PROFORMA FOR "MADHUMEGAM"

Ward No	:	Nationality	:
IP NO	:	Religion	:
Bed No	:	Occupation	:
Name	:	Income	:
Age	:	Date of admission	:
Sex	:	Date of discharge	:
Permanent Address	:	Diagnosis	:

Medical Officer:

Temporary Address:

Government Siddha Medical College,
Chennai-600 106

Education Status:

COMPLAINTS AND DURATION :

HISTORY OF PRESENT ILLNESS :

HISTORY OF PREVIOUS ILLNESS :

PERSONAL HISTORY

PERSONAL HABITS

FAMILY HISTORY :

SIDDHA ASPECT

NILAM

Kurinchi (Mountain and their adjoining areas)

Mullai (Forest and their adjoining areas)

Marutham (Fertile and their adjoining areas)

Neithal, (Sea and their adjoining areas)

Palai (Desert and their adjoining areas)

PARUVAKAALAM

Kaar Kaalam (Avani – Purattasi)

Koothir kaalam (Iyppasi – Karthigai)

Munpani Kaalam (Margazhi – Thai)

Pinpani Kaalam (Masi – Panguni)

Elavenil Kaalam (Chitthirai – Vaikasi)

Muthuvenil Kaalam (Aani – Aadi)

YAAKAI (UDAL)

Vali Udal

Azhal Udal

Iya Udal

Kalappu Udal

GUNAM

Sathuva gunam

Rajo gunam

Thamo gunam

PORI (Gnanenthirium) PULANGAL

Mei	-	Unarthal
Vai	-	Suvaithal
Kan	-	Parthal
Mooku	-	Mugarthal
Sevi	-	Kettal

KANMENTHIRIYAM / KANMAVIDAYAM

Kai	-	Koduthal
Kal	-	Nadathal
Vai	-	Paesal
Eruvai	-	Malam Kalaithal
Karuvai	-	Anandhithal

UTKAYAM – ATHAKAYAM

Puyam

Sayam

Kaal

Patham

PIRA URUPPUKALIN NILAI

Iruthayam

Puppusam

Eraipai

Kalleeral

Manneeral

Kudal

Siruneeragam

Karuppai

Moolai

UYIR THATHUKKAL

VATHAM

Piranan

Abanan

Viyanan

Udhanan

Samanan

Nagan

Koorman

Kirukaran

Devathathan

Thananjeyan

PITHAM

Analapitham

Ranjagappitham

Alsagapitham

Saathagapitham

Pirasaga pitham

KAPHAM

Avalambagam

Kilethagam

Pothagam

Tharpagam

Santhigam

UDAL THATHUKKAL

Saaram

Senner

Oon

Kozhuppu

Enbu

Moolai

Sukkilam / Suronitham

ENVAGAI THERVUGAL

NAA Niram
Thanmai
Pulan

NIRAM

MOZHI Thazhntholi
Uratholi

VIZHI Niram
Thanmai
Pulan

SPARISAM

MALAM

Niram
Erugal / Elagal
Manam
Nurai

MOOTHIRAM

NEERKURI:
Niram
Edai
Manam
Nurai
Enjal

NEIKURI

NAADI

Thani Naadi
Thontha Naadi
Mukkutra Naadi
Thoda Naadi

MODERN ASPECT

GENERAL EXAMINATION:

Consciousness

Body build

Anaemia

Jaundice

Cynosis

J.V.P.

Clubbing

Tracheal deviation

Abdominal distention

Pedal oedema

Lymphadenopathy

VITAL SIGNS

Temperature:

Pulse :

Rate
Rhythm
Volume
Character

Respiration Rate:

Heart Rate :

Blood Pressure :

Height :

Weight :

INVESTIGATION:

1. Urine
   - Albumin
   - Sugar
   - Deposit
   - Creatinine
   - Ketones

2. Motion
   - Ova
   - Cyst

3. Blood
   - TC
   - DC
   - ESR
   - Hb%
   - Group
   - Urea
   - Creatinine
   - Total Cholesterol

4. Blood Sugar
   - Random
   - Fasting
   - Post Parandial

5. Oral glucose Tolerance Test (GTT)

   Blood Sugar : Fasting
   ½ hour
   1 hour
   1 ½ hour
   2 hour

6. Glycosylated Haemiglobin

7. X-Ray Chest

8. ECG

9. Eye – fundus examination

S.No.	Before Treatment	DOA	14th Day	24th Day	28th Day	35nd Day	45th Day
1.	Polyuria						
2.	Poly phagia						
3.	Poly dipsia						
4.	Glycosuria						
5.	Balanitis / Pruritis Vulvae						
6.	Itching all over the body						
7.	Pain all over the body						
8.	Dryness of the mouth and throat						
9.	Constipation						
10.	Skin infection						
11.	Emaciation						
12.	Dry Skin						
13.	Peripheral Neuritis						
14.	Diabetic foot ulcer						

DATE	DAILY REPORT	MEDICINE
ADVICE: **MEDICAL OFFICER**		

DISCHARGE SHEET

Government Siddha Medical College,
Post-Graduate Department,
Pothu Maruthuvam Branch,
Chennai -600106.

PROFORMA FOR "MADHUMEGAM"

Discharge Summary

<table>
<tr><td rowspan="6">I.P.No:</td><td>Name:</td></tr>
<tr><td>Age/Sex:</td></tr>
<tr><td>Occupation:</td></tr>
<tr><td>Income:</td></tr>
<tr><td>Nationality:</td></tr>
<tr><td>Religion:</td></tr>
<tr><td>Date of Admission:

No of days treated:

DIAGNOSIS:

RESULT:</td><td>Date of Discharge:

MEDICAL OFFICER</td></tr>
</table>

PATIENT CONDITION

Signs and Symptoms	On Admission	On Discharge
Polyuria		
Polyphagia		
Polydipsia		
Glycosuria		
Pruritis Vulvae		
Itching all over the body		
Pain all over the body		
Dryness of the mouth and throat		
Constipation		
Skin infection		
Emaciation		
Dry Skin		
Peripheral Neuritis		
Diabetic Foot ulcer		

Pulse :
Weight :
Blood Pressure :
Blood Sugar :
Urea :
Serum Cholesterol :
Urine :

- Albumin :
- Sugar :
- Deposits :
- Ketone bodies :

OBSERVATIONS OF CASES

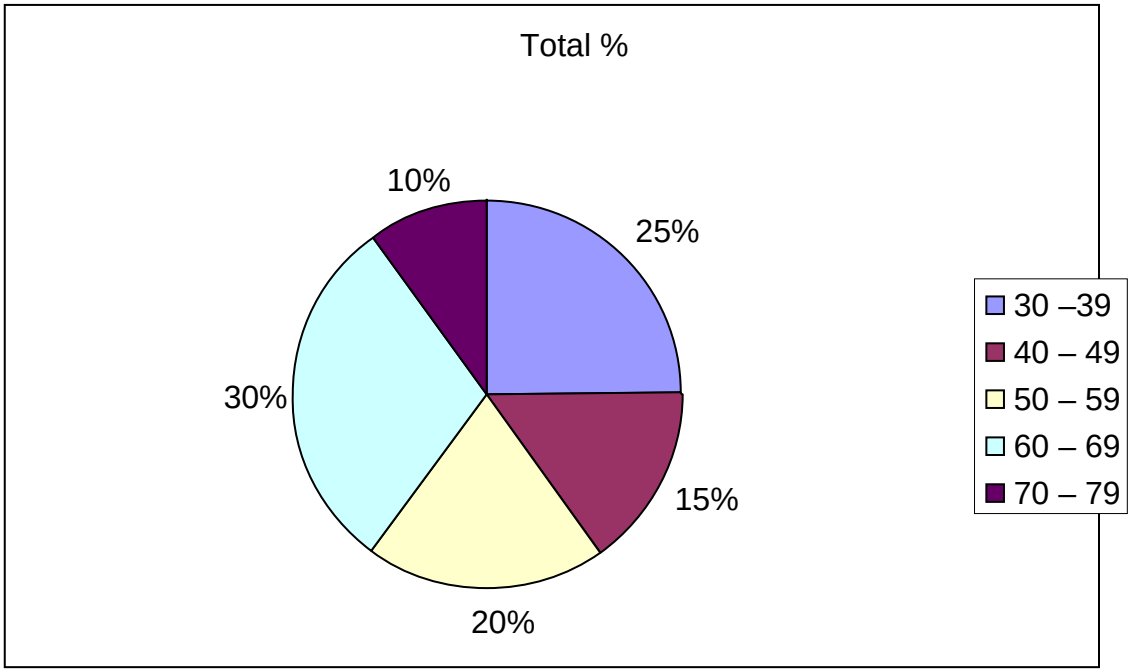
The factors considered for observation for the purpose of the study comprised of the following:

- Age – wise / gender – wise
- Gender – wise classification.
- Distribution of thinai
- Paruvakaalam [Seasonal incidence]
- Nature of job
- Socio economic status
- Dietary habits
- Family history
- Body weight
- Classification of results according to vali, Azhal and Iyam
- Ezhu Udal Kattugal
- Enn vagai theervugal
- Classification on the basis of Neikuri
- Naadi
- Signs and Symptoms.
- **Urine Sugar**
    - Fasting
    - Post Prandial
- **Blood Sugar**
    - Fasting
    - Post Prandial
- Efficacy of Medicine
- Bio Chemical Analysis of the trial drug
- Toxicological study of the trial drug
- Pharmacological studies of the trial drug

TABLE 1

AGE – WISE / GENDER WISE ANALYSIS

Age	Male	Male %	Female	Female %	Total	Total %
30 –39	2	10	3	15	5	25
40 – 49	1	5	2	10	3	15
50 – 59	1	5	3	15	4	20
60 – 69	3	15	3	15	6	30
70 – 79	1	5	1	5	2	10

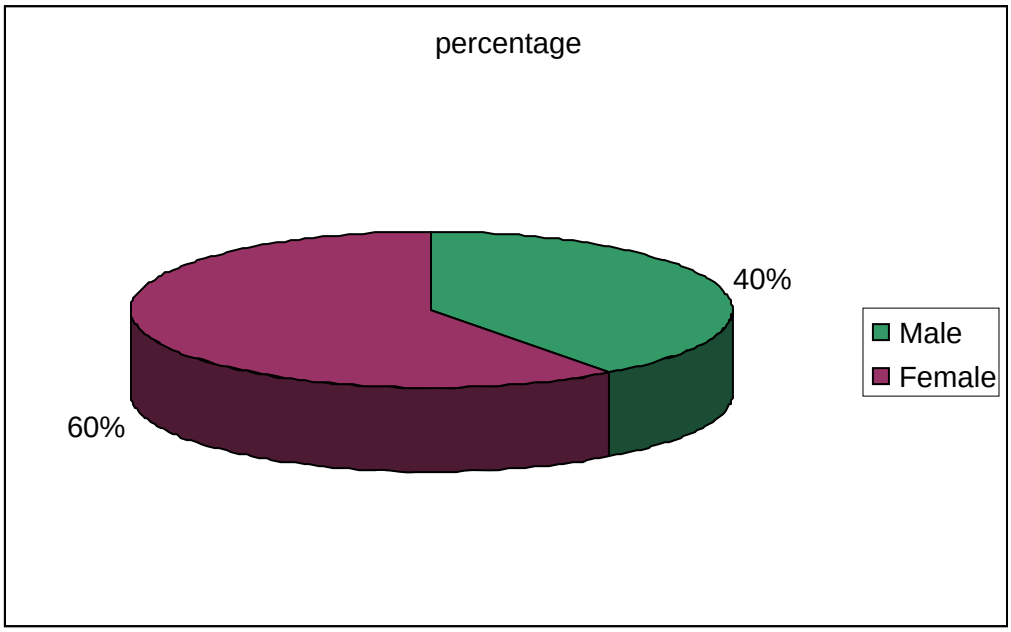


It is observed from the above analysis that the incidence of Madhumegam is more in the age group of 60-69 in males and 60 – 69, 50 – 59, 30 – 39 in females. On the age wise analysis as given in the above chart it may be noted that the incidence of the diserse is more in the age group of 60-69 and 30 – 39 which is 30% and 25% in each of these groups.

TABLE 2

GENDER-WISE CLASSIFICATION

SL.NO	Gender	No of patients	percentage
1	Male	8	40
2	Female	12	60

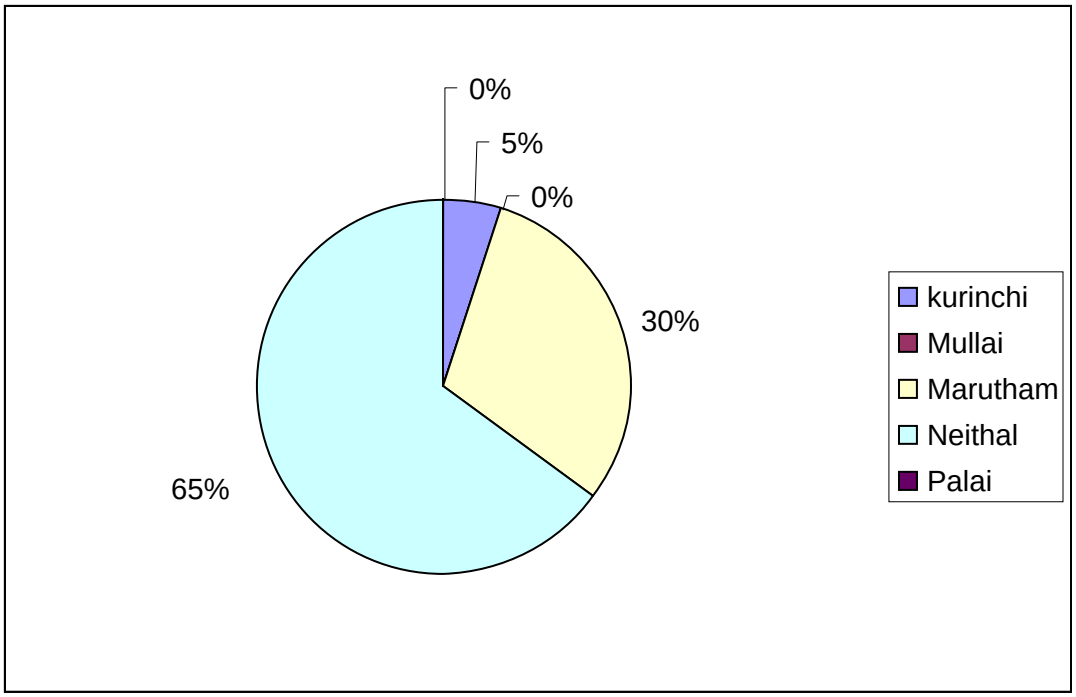


The Study done in the basis of patients who came for treatment of madhumegam, at the hospital reveals that majority of them are females.

TABLE 3

DISTRIBUTION OF THINAI

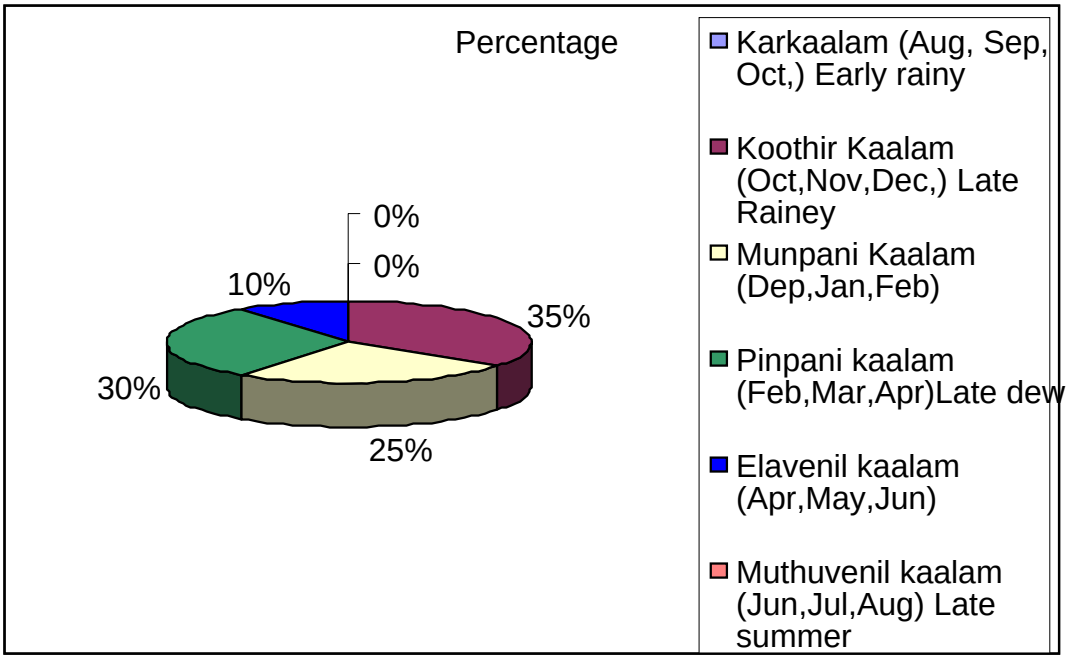
Thinai	No of cases	Percentage
kurinchi	1	5
Mullai	0	0
Marutham	6	30
Neithal	13	65
Palai	0	0



From the chart as above it may observed that people living in Neithal are prone to Madhumegam while compared to others. This is very evident from the fact that 65% of the patients with diabetes are from this region.

TABLE 4
SEASONAL INCOME (PARUVAKAALAM)

Paruvakaalam	Months	No. of cases	Percentage
Karkaalam (Aug, Sep, Oct,) Early rainy	Aavani /purattasi	0	0
Koothir Kaalam (Oct,Nov,Dec,) Late Rainey	Ayppasi/ Karthigai	7	35
Munpani Kaalam (Dep,Jan,Feb)	Margazhi/t\Thai	5	25
Pinpani kaalam (Feb,Mar,Apr)Late dew	Masi/panguni	6	30
Elavenil kaalam (Apr,May,Jun)	Chittirai/Vaigasi	2	10
Muthuvenil kaalam (Jun,Jul,Aug) Late summer	Aani/Aadi	0	0

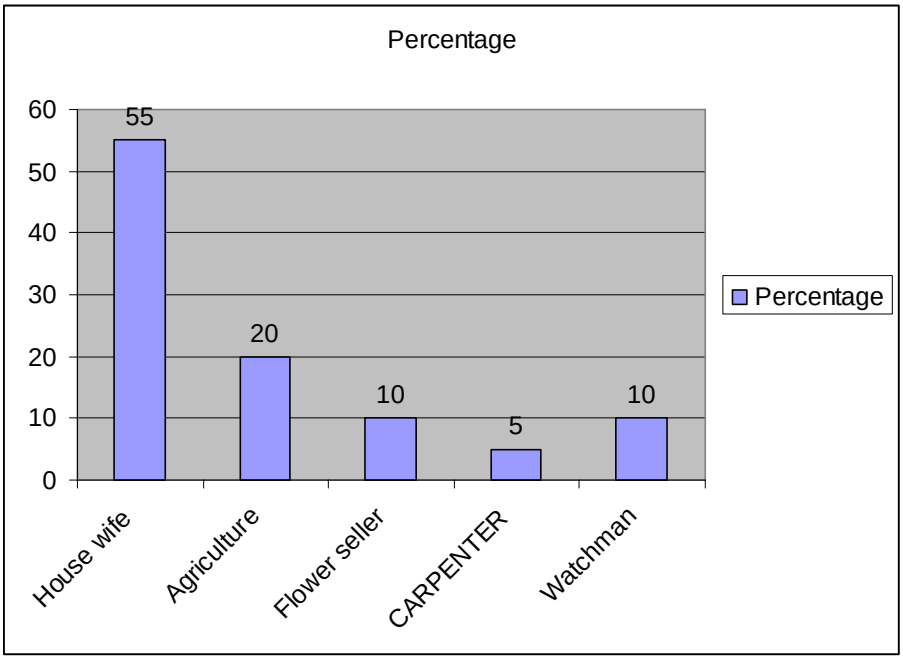


The chart clearly indicates that seasonal variances do not have a serious impact. In almost throughout the year, Madhumegam sets in.

TABLE 5

NATURE OF JOB

Occupation	No. of patients	Percentage
House wife	11	55
Agriculture	4	20
Flower seller	2	10
Carpenter	1	5
Watchman	2	10

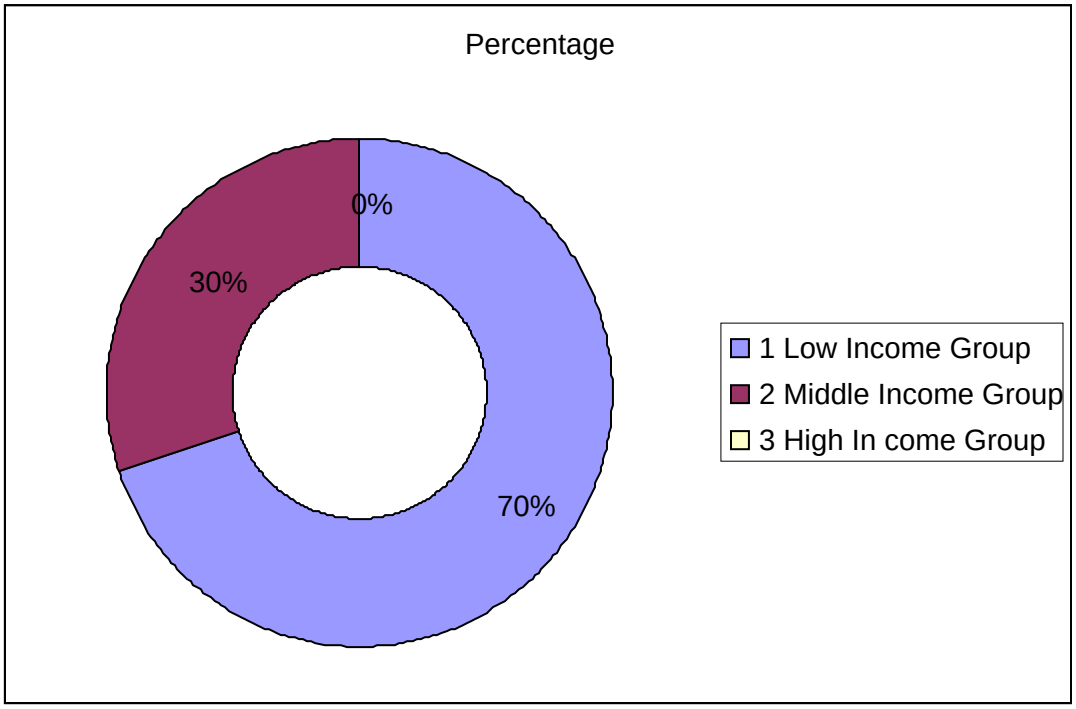


From the above figures it may be noted that the incidence of Madhumegam is more in house wives i.e. 55% of total patients and not on the labour community.

TABLE 6

SOCIO ECONOMICS STATUS

SL.NO	In COME	No. of patients	Percentage
1	Low Income Group	14	70
2	Middle Income Group	6	30
3	High In come Group	0	0

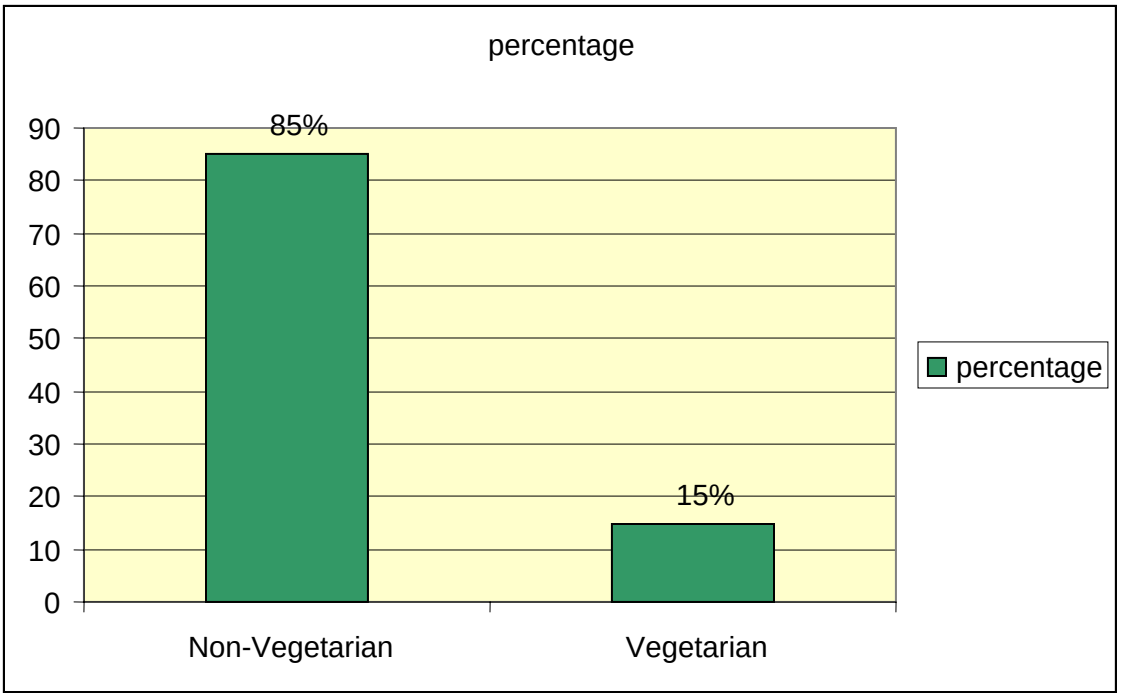


70% of the people to low income group, which shows Madhumegam which once was considered to be the disease of rich affects poor also.

TABLE 7

DIETERY HABITS

Dietary Habits	No. of cases	percentage
Non-Vegetarian	17	85
Vegetarian	3	15

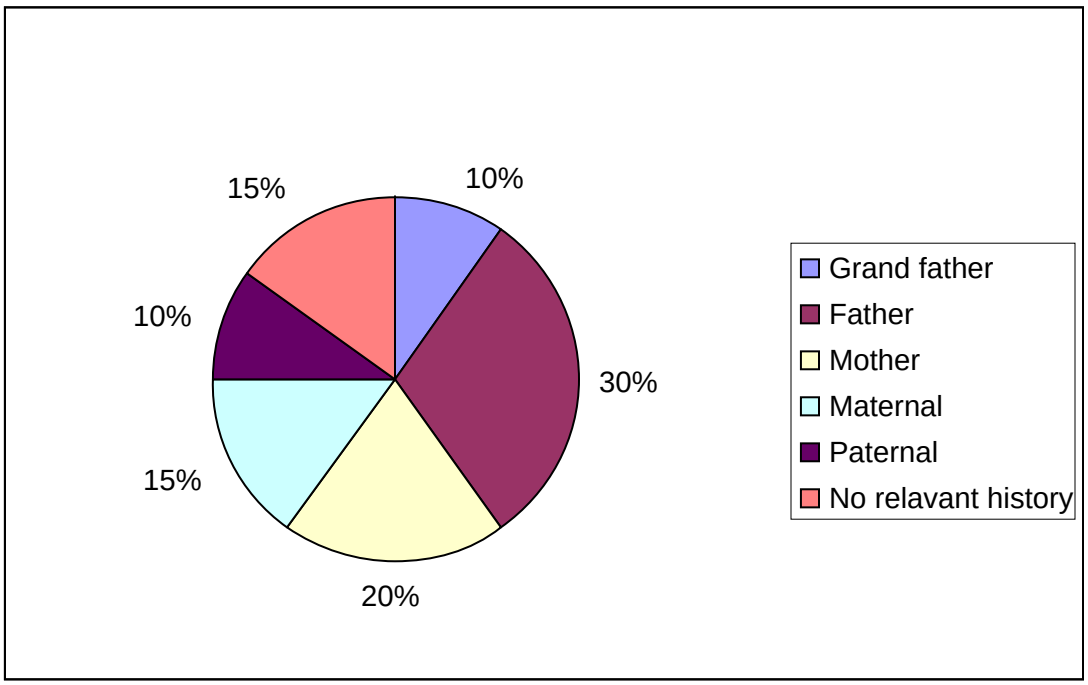


The figures that were analysed as above taking in to account the dietary habits clearly indicates that the incidence of Madhumegam is lagely on people who are non-vegetarians i.e. 85%.

TABLE 8

FAMILY HISTORY

Family History	No. of cases	percentage
Grand father	2	10
Father	6	30
Mother	4	20
Maternal	3	15
Paternal	2	10
No relavant history	3	15

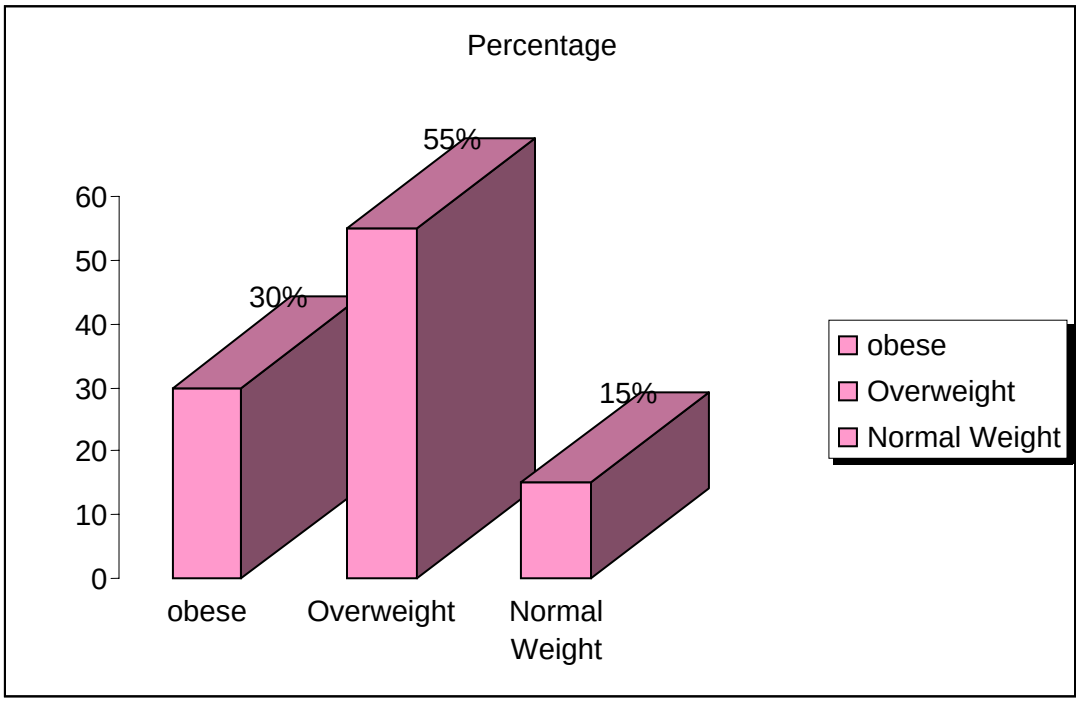


The study taken up on the basis of patients background gives a picture that family history i.e., hereditary factors is significant in madhumegam. While analyzing the facts still deeper, it is noted that parental incidence is much higher for the onset of the disease. However it is not out of place to mention that even in cases where the grand parents had diabetes it passes on to the next generations also.

TABLE 9

BODY WEIGHT

Weight	No, of cases	Percentage
obese	6	30
Overweight	11	55
Normal Weight	3	15

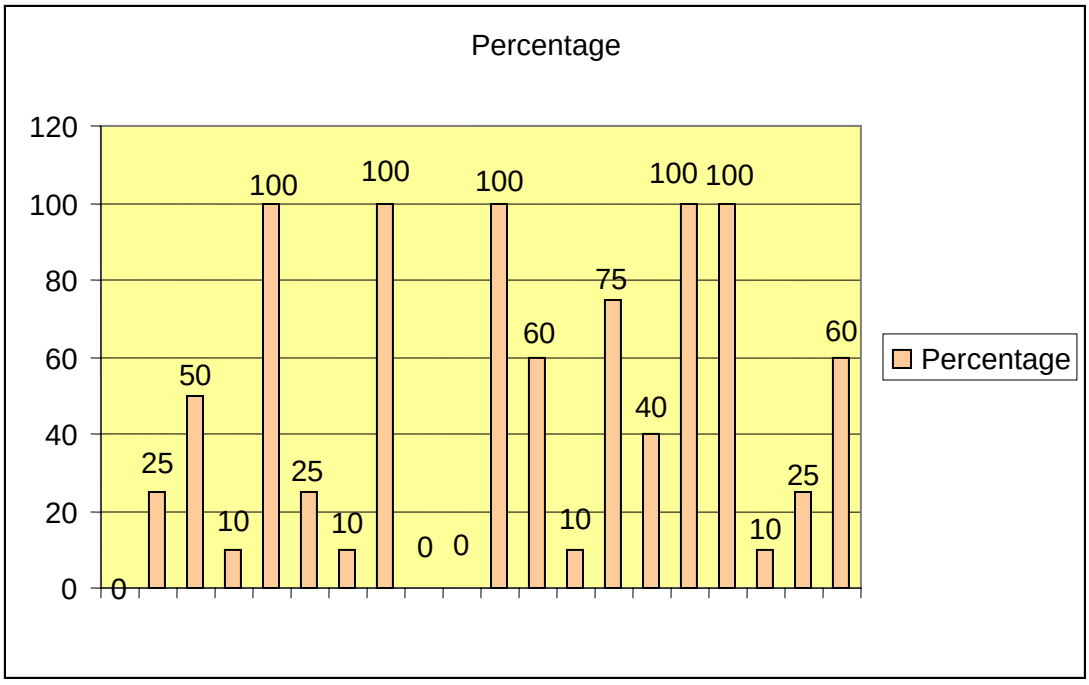


The analysis of the above data clearly indicates that people with over. Weight body structure, are more prone to Madhumegam. This clearly indicates that the body weight is more relevant for the incidence of the disease. Even people with normal weight are also affected.

TABLE-10

CLASSIFICATIONS OF RESULTS ACCORDING TO VALI, AZHAL AND IYAM

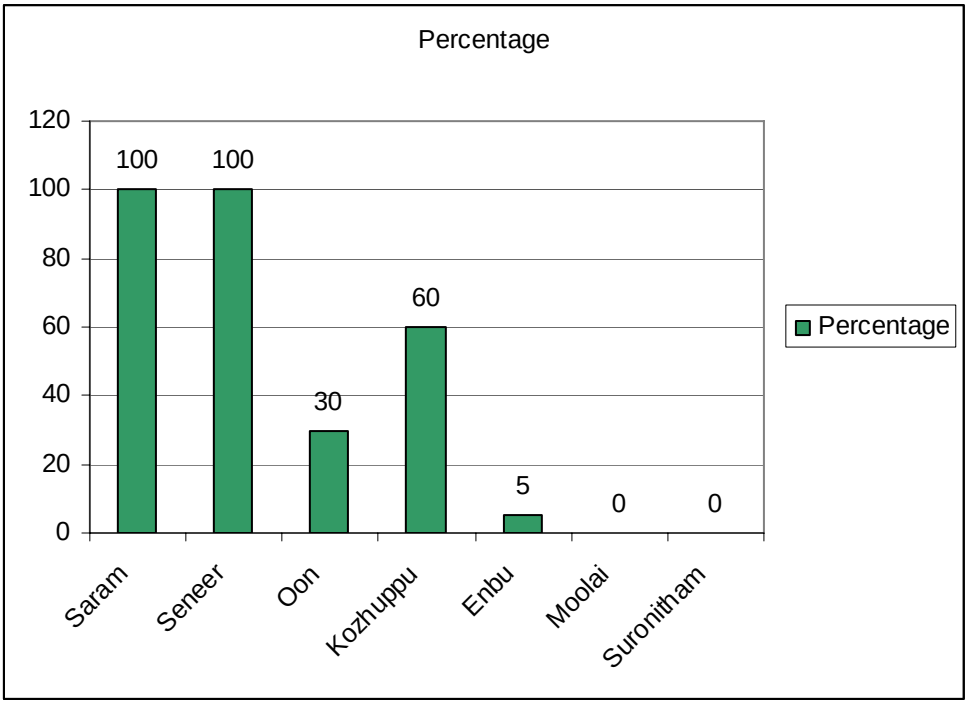
	PARTICULARS	NO,OF CASES	Percentage
VALI	Piranan	0	0
	Abanan	5	25
	Vijanan	10	50
	Udanan	2	10
	Samanan	20	100
	Nagan	5	25
	Koorman	2	10
	Kirukaran	20	100
	Devathathan	0	0
	Thananjeyan	0	0
	Anala pitham	20	100
AZHAL	Ranjaga pitham	12	60
	Alosaga Pitham	2	10
	Saathaga pitham	15	75
	Pirasaga pitham	8	40
IYAM	Avalambagam	20	100
	Kilethaam	20	100
	pothagam	2	10
	Tharpagam	5	25
	Senthigam	12	60



In vali Samanan and Kirukaran in Azhal Anala pitham and in Iyam Avalambagam and Kilethaam are affected in all patients i.e. 100%

TABLE-11
EZHU UDAL KATTUGAL

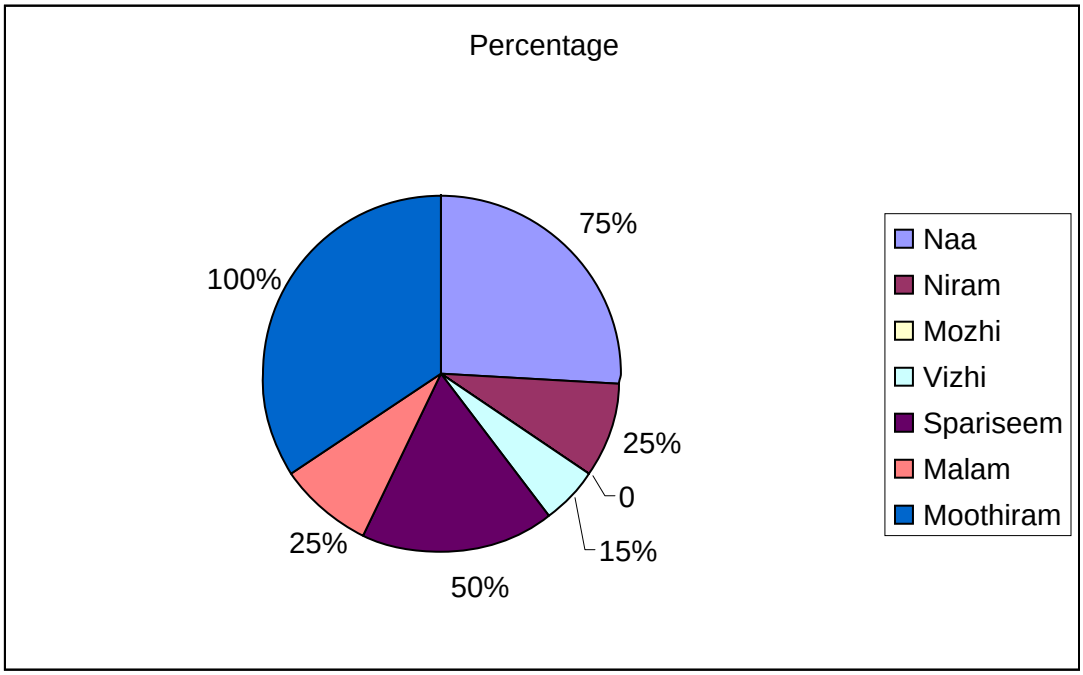
EZHU UDAL KATTUGAL	No. of Cases	Percentage
Saram	20	100
Seneer	20	100
Oon	6	30
Kozhuppu	12	60
Enbu	1	5
Moolai	0	0
Suronitham	0	0



From the above chart we can observe that Saram and Senner and affected in all patients i.e. 100%. Oon, Kozhuppu are affected to the extend of 30% and 60% respectively.

TABLE-12
ENNVAGAI THERVUGAL TREATNENT -BEFORE

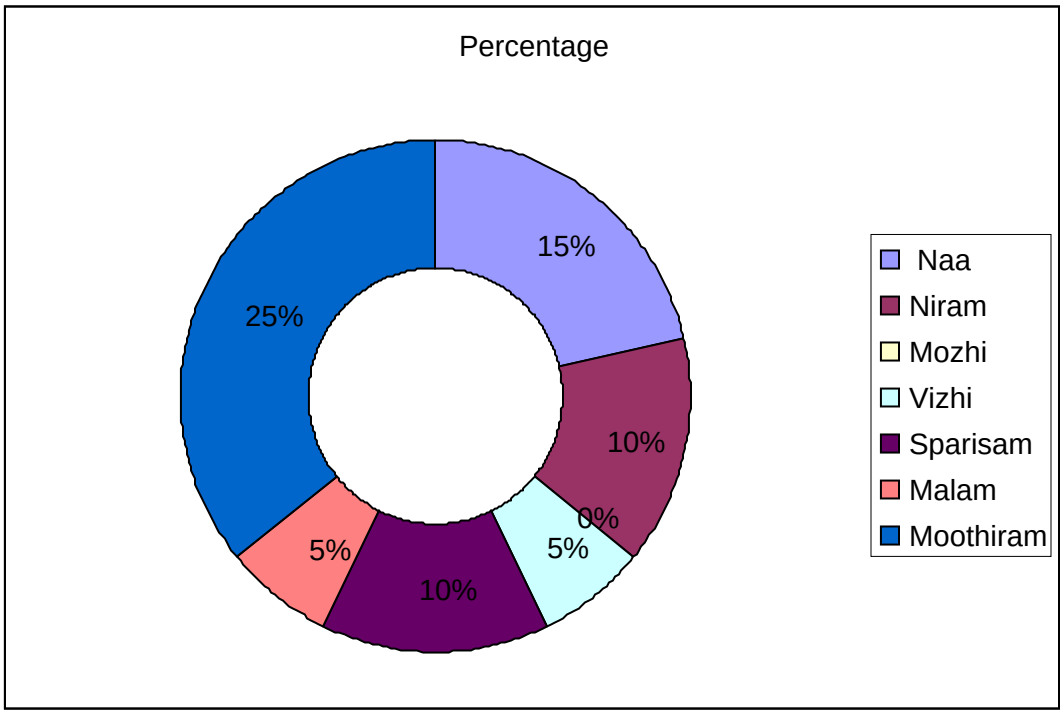
Thervugal	No. of cases	Percentage
Naa	15	75
Niram	5	25
Mozhi	0	0
Vizhi	3	15
Spariseem	10	50
Malam	5	25
Moothiram	20	100



On anlysing the facts on diagnosis Moothiram had 100% impact and Naa had 75% impact. It may further be noted from the figures that Mozhi is not at all affected by the disease.

TABLE-13
ENN VAGAI THERVUGAL-AFTER TREATMENT

Thervugal	No.of cases	Percentage
Naa	3	15
Niram	2	10
Mozhi	0	0
Vizhi	1	5
Sparisam	2	10
Malam	1	5
Moothiram	5	25

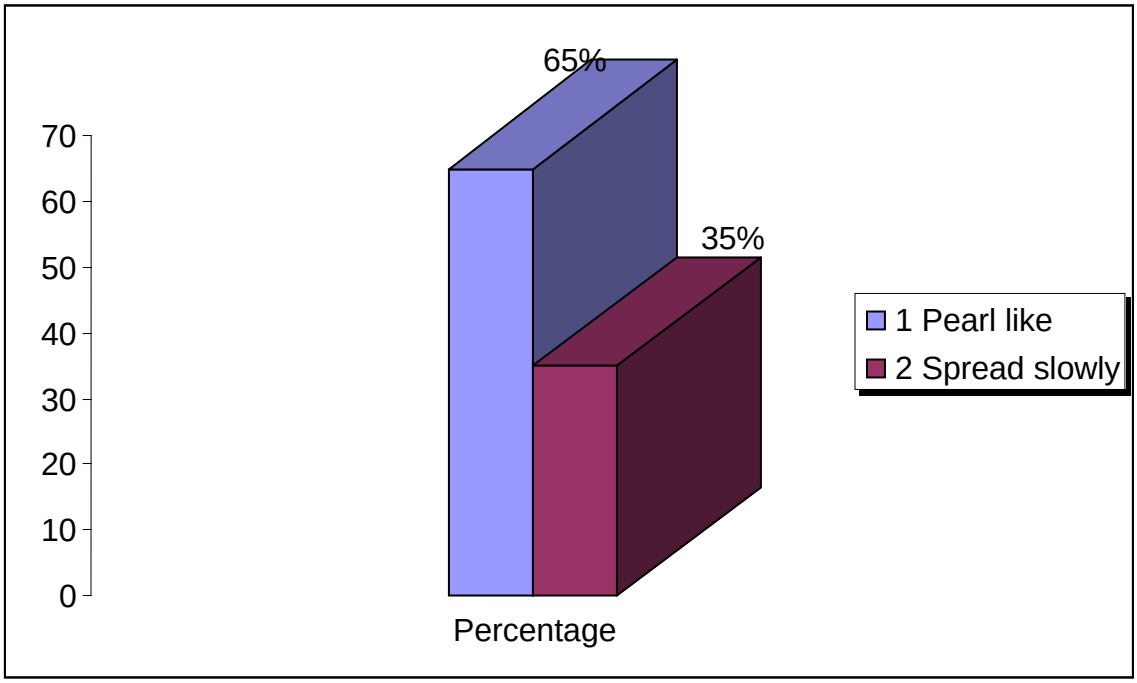


From the study it may be noted that after treatment, there is a definite change, which is evident from the fact that Moothiram had only 25% impact and others it is almost negligible.

TABLE-14

CLASSIFICATION ON THE BASIS OF NEIKVRI

SL.NO	Neikuri	No.of cases	Percentage
1	Pearl like	13	65
2	Spread slowly	7	35



In respect of 65% of the patients when oil is dropped in the urine it looks like pearl, which indicates Iyam is prominent. In the balance 35% it spreads slowly.

TABLE-15

SIGNS AND SYMPTOMS

Signs and symptoms	No. of cases Before Treatment	Percentage	No. of cases after treatment	Percentage
Polyuria	20	100	5	25
Polyphagia	20	100	2	10
Polydipsia	20	100	2	10
Glycosuria	20	100	4	20
Pruuitis vulvae	3	15	0	0
Itching all over the body	4	20	0	0
Pain all over the body	20	100	5	25
Dryness of the month and throat	10	50	0	0
Constipation	8	40	0	0
Skin infection	2	10	0	0
Emaciation	20	100	2	10
Dry skin	10	50	0	0
Peripheral Neuritis	8	40	3	15
Diabetic Foot ulcer	2	10	0	0

In respect of the patients with Madhumegam the clinical symptoms of polyuria, polyphagia, glycosuria pain all over the body and Eonaciation were present in all cases i.e., 100% before treatment.

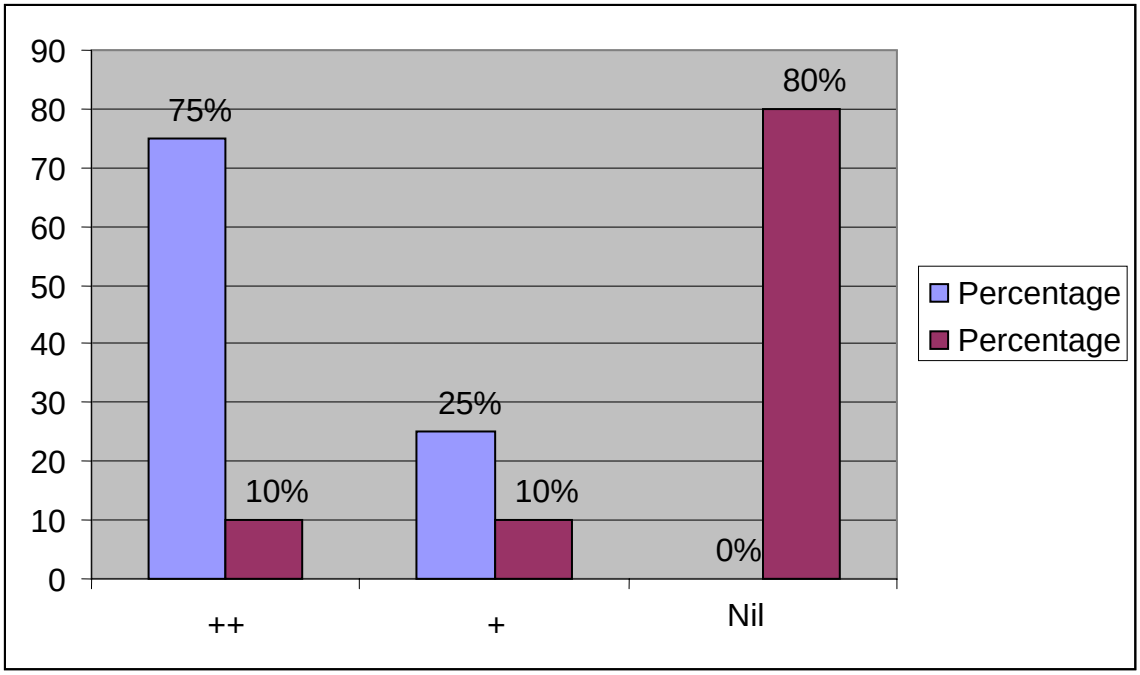
The clinical signs and symptoms were improved after treatment showing only 25% of the people have polyuria and pain all over the body. 20%

of the people have glycosuria and 10% of the people have polyphagia polydipsia and emaciation.

TABLE-16

URINE SUGAR – FASTING

Urine	Before Treatment	Percentage	Before after treatment	Percentage
++	15	75	2	10
+	5	25	2	10
Nil	0	0	16	80

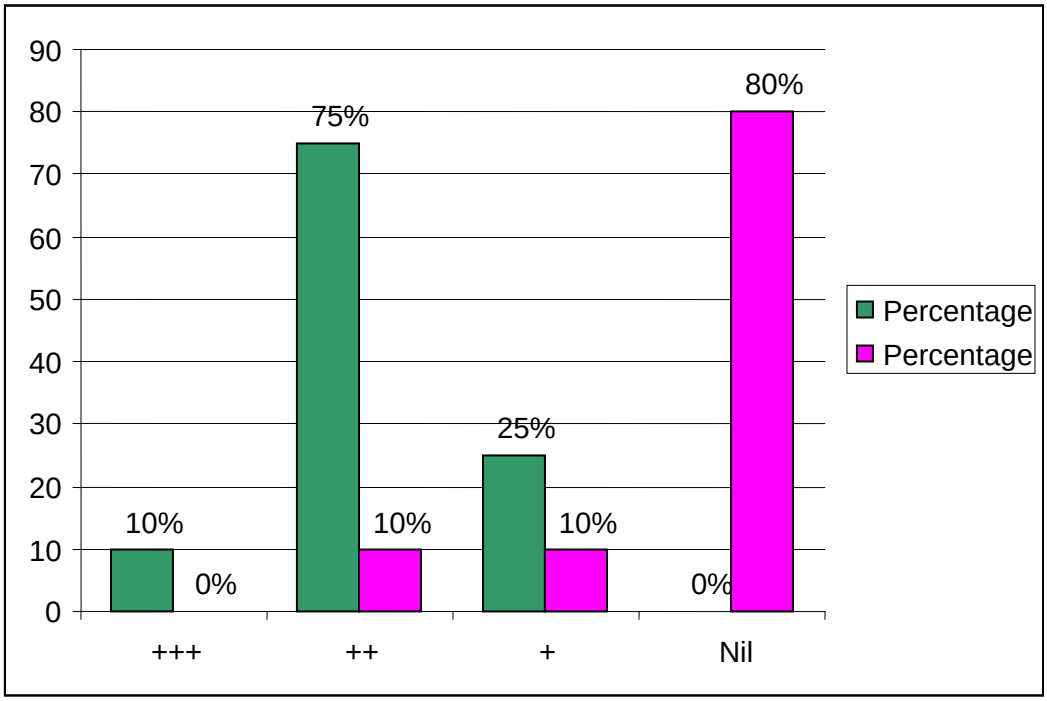


From the above chart it may be observed that the urine sugar position on fasting, after treatment had drastically. It was “Nil” in 80%of the cases after treatment.

TABLE-17

URINE SUGAR-POST PRANDIAL

Urine - Sugar	Before Treatment	Percentage	After Treatment	Percentage
+++	2	10	0	0
++	13	75	2	10
+	5	25	2	10
Nil	0	0	16	80

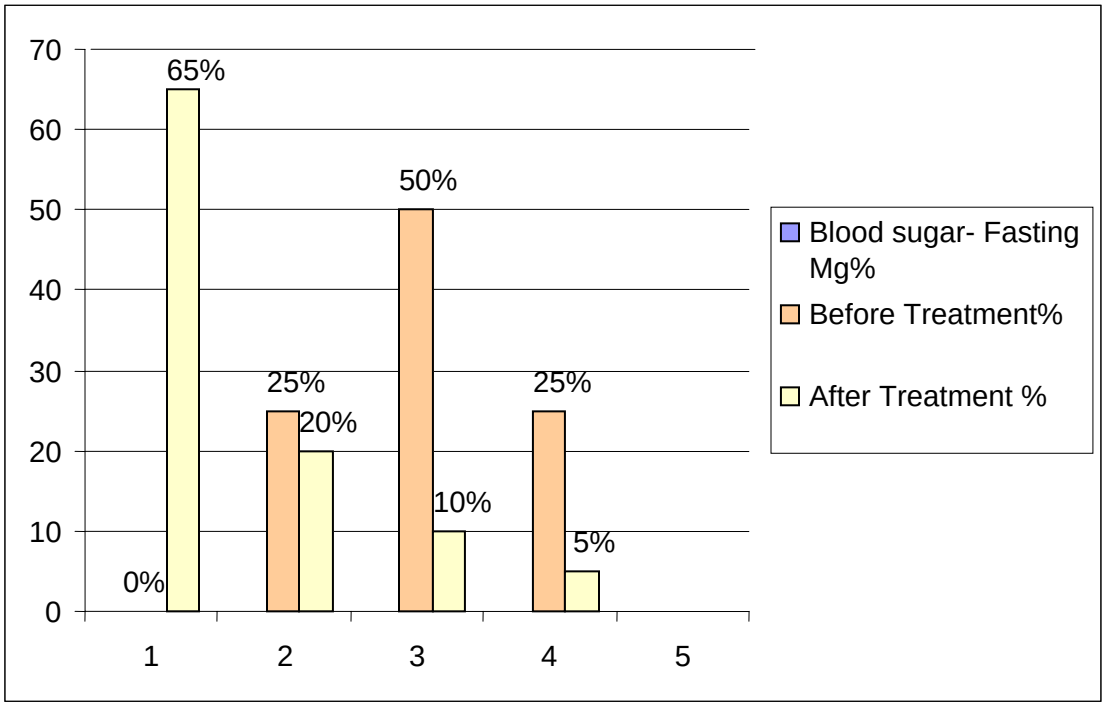


It may be noted that the post prandial urine sugar position, after treatment had improved drastically. It was "Nil" in 80% of the cases, after treatment.

TABLE-18

BLOOD SUGAR- FASTING

Blood sugar- Fasting Mg%	Before Treatment	Before Treatment%	After Treatment	After Treatment %
60-109	0	0	13	65
110-129	5	25	4	20
130-149	10	50	2	10
Above 150	5	25	1	5

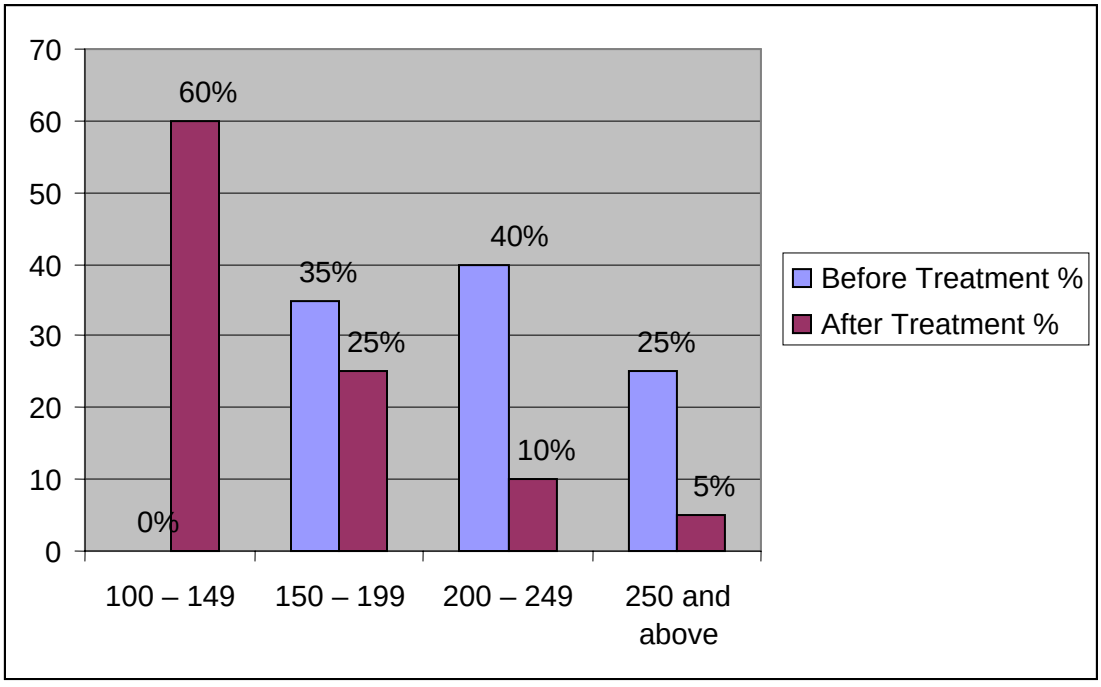


Fasting blood sugar has improved in 65% of the cases.

TABLE 19

BLOOD SUGAR POST – PRANDIAL

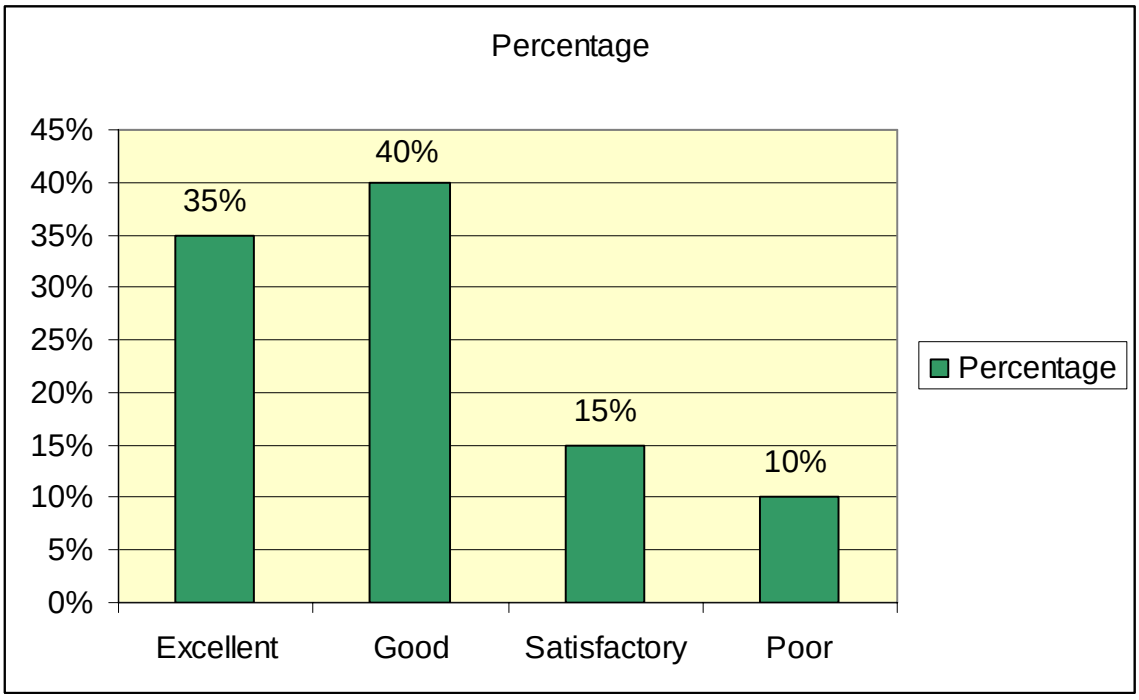
Blood Sugar PP – mg%	Before Treatment	Before Treatment %	After Treatment	After Treatment %
100 – 149	0	0	12	60
150 – 199	7	35	5	25
200 – 249	8	40	2	10
250 and above	5	25	1	5



The blood sugar psot prandial has become normal for 60% of the cases.

TABLE 20
EFFICACY OF MEDICINE

S.No.	Final Results	No. of Cases	Percentage
1.	Excellent	7	35 %
2.	Good	8	40 %
3.	Satisfactory	3	15 %
4.	Poor	2	10 %



The results shows Excellent in 35% of the cases. Good in 40% of the cases. Satisfactory in. 15% of the cases. Poor in 10% of the cases.

BIOSTATISTICS

HYPOTHESIS I: NUMBER OF PATIENTS CURED ITS MORE THAN NOT CURED

Among the total number of Inpatients treated for diabetes during the study period, the sampled 20 patients, namely both male and female have been classified under two categories as (i) cured and (ii) not cured. The following table shows the division of both cured and non-cured male and female patients.

Table I: Effectiveness of Treatment for diabetes

No. of Patients cured		Ratio of Male to Female	No. of patients not cured		
M	**F**	**M : F**	**M**	**F**	**Ratio of M to F**
7	8	1 : 1.14	2	3	1: 1.5

to female under cured and not cured categories is 1:1.4 and 1:1.5 respectively. It is further observed that in both the cases the ratio of female is greater than male patients. In general, out of the 20 sampled patients, 15 are found to be cured ad 5 are not cured where the ratio of cure to not cured remains 3:1. This means that, irrespective of gender, out of 4 patients treated, 3 get cured and 1 does not. From this table, it is inferred that the **treatment is good for diabetes ad hence the first hypothesis of the present study is proud.**

Hypothesis : II The inpatients treated for diabetes have been benefited by the services the Siddha Hospital.

In order to verify the second hypothsis of the present study, an opinion survey was conducted and the feed back from the patients were recorded. Accordingly, the opinion of the patients have been classified in to four as (i) Excellent (ii) Good (iii) Satisfactory ad (iv) Poor. The second hypothesis is

verified with the help of the famous statistical tool known as Chi-Square Test of Goodness of fit. The observed values have been recorded from which the table of expected frequencies has been formulated.

Opinion about treatment – Table II of observed frequencies

Opinion	Male	Female	Total
Excellent	4	3	7
Good	4	4	8
Satisfactory	1	2	3
Poor	1	1	2

Table of Expected frequencies

Opinion	Male	Female
Excellent	3.5	3.5
Good	4	4
Satisfactory	1.5	1.5
Poor	1	1

χ^2 value is calculated with the help of the formula.

$$\chi^2 = \frac{\Sigma (0\text{-}E)^2}{\Sigma E} = \frac{1}{20} = 0.05$$

The table value of χ^2 at 5% significant level and for 3 degrees of freedom is found to be 7.81. **Since the calculated value of χ^2, namely 0.05 is less than the table value, the second hypothesis is accepted.** Therefore, it is concluded that **the Inpatients treated for diabetes have been benefited by the services of the Siddha Hospital.**

DISCUSSION

The drastic change in the life style and the food habits of people in the modern world has definitely played a very important role in the health aspect of humanity. Moreover the present day environment also has contributed significantly in the increase of health hazards. The disease Madhumegam, which was once supposed to be associated with the rich community has spread it's wings to be entire humankind. It is not only the elders who are affected but today it starts in childhood also. The way it is exploding has made the medical practitioners and researchers to ponder over the issue and analyse the causes and impact of the same in different angles. In this study, The various factors were taken in to consideration with the small group of twenty patients. The aspects that were looked in to for the study are discussed as follows:

Age wise analysis:

The age wise classification shows that the Madhumegam is common in the age group above 50 years. Usually the non-insulin dependent diabetes mellitus occurs only in the age group of above 50 years. In India above the age of 60 years 16.5% of urban and 5.3% of rural population show diabetics mellitus.

- International diabetic Monitor

Gender wise:

Above classification 2 shows female gender are affected more. Recent studies show that more women have diabetes compared to men. Diabetes in women is also more worrying for the following reasons.

Women are more at risk of developing certain complication of diabetes like reduced blood flow to the feet and heart attack.

Even more alarming is the development of so called "Gestational diabetes" for this is a big risk factor for the child developing diabetes in the

future. More over women do not have that much of manual labour while compared to man.

Thinai (Land)

People living in Neithal are prove to Madhumegam. Because the land is near sea and the food crops grown in that are is Alkaline. Moreover Neithal land makes people obese which cause the disease.

(Siddha Maruthuvanga Churukkam P-256)

Liver the major site of metabolism is affected for the people living in Neithal land i.e. Hepatomegaly is more common so, Madhumegam is more common Neithal land. Chennai the Diabetic capital of India is a Neithal land.

beŒjåš .. ..

tšYW¥ig Å¡F«

- nehæšyh be¿ g¡f« 3

Seasonal Incidence:

The chart clearly shows that seasonal variances do not have a serious impact on the disease Madhumegam.

The incidence is more in Mathuvenil Kaalam in which the body is weak and dry due to excessive heat. This period is also called Anatha Kaalam.

(Siddha Maruthuvanga Churukkam Page-292).

Nature of Job:

Incidence of Madhumegam is more in housewives. Now a days due to modernization and invention of electrical and electronic kitchen equipment the women lack physical exercise hence more prone to Madhumegam it was found that most diabetes were on manual occupations than non diabetes people.

- A. Park's Social and Preventive medicine.

Socio Economic Status:

People belonging to the lower income group are affected by Madhumegam. Recent research indicates that the poor were also prone to diabetes. Reserch was being conducted to analyse whether rapid changes in their life style or the stress of poverty triggered diabetes.

Dietary Habits:

Madhumegam incidence is more among non vegetarians. Further it could also be noted that, people who are used to fast food and fried food are more prone to diabetes as they have more calories of fat.

To Quote from Yugi:

" c‰gé¡F« ghšb eŒahèiw¢Á fŸshš

As per Agasthiyar

bfhG¤j Ûåiw¢Á nghij

xJ ÚçêÎ nru"

- mf¤Âa® 1200

Family History:

Genetics plays an important role in Madhumegam.

To quote from Thirumoolar Karukidai Vaitheyam.

"Jiw nf£»‹ f®¥g¤Âš

Jt§»a nkf§ nfŸ"

Body weight:

In obese and over weight people the incidence of Madhumegam is more. The body mass index (BMI) in adult are derived from the formula, weight (kg)/height (m)2. The acceptable normal range of BMI is 20 to 25.

The BMI between 25.0 to 29.9 are classified as overweight. Obesity is taken to strt at a BMI of 30.0 to 39.9.

(Davidsons Text book of Medicine Page -526).

As per Yugi, obesity is among the cause of diabetes.

“j‰gé¡FŠ rßuªjh‹ äf¥ gU¡fš”

-ô» it¤Âa Áªjhkâ

UYIR THATHUKKAL

In vali Samanan and Kirukaran are affected. The action of Samanan is to help in digestion and absorption of food and streng then the body.

- P.146 Siddha Maruthuvanga Churkkam

If Samanan is affected. The digestive system gets affected which in turn affect the metabolison of carbohydrates protein and fat.

Kiruaran maintains the saliva secretion and causes appetite. If deranged it cause excessive appetite (Poly Phagia) which is one of the symptoms of madhumegam.

In Azhal, Anala Pitham is affected greatly in all patients. In general Analapitham action is exactly between the stomach and small intestine which means the pancreatic action is mainly maintained by Analapitham. If derangement causes the disease madhumegam.

In Iyam Avalambagam and kilethagam are affected. The action of Avalambagam makes the other four Iyam to the balance. The imbalance in any one of Kilethegam makes the Avalambagam affected.

The function of Kilethegam is to make the contents of the stomach ready for digestive process. If the function get deranged the initial phage of metabolism gets affected.

Ezhu Udal Kattugal:

In madhumegam saram and seneer are affected in all patients. Affected saram makes the patient emaciated and loss of interest in general activities in saram decreased state causes dryness of the skin.

Affected seneer, makes nervousness, dryness and diministion of the colour of the skin. These in turn affect the other udal thathugal also.

Enn Vagai Thervugal

Enn vagai thervugal the main diagnostic principle shows.

Naa is affected in 75%, which is dry

Niram is affected in 25%

Mozhi is not affected

Vizhi is affected in 15%

Sparisam is affected in 50%, which causes dry skin and peripheral neuritis

Malam is affected in 25% who are constipated

Moothiram is affected 100% all patients who has polyuria

After Treatment

Naa improved well in the patient

Niram is affected in only 10%

Mozhi is not affected

Vizhi is affected in 10%

Sparisam is affected in 10%

Malam is affected in 5%

Moothiram is affected in 25%

Neikuri

When oil is dropped in urine it looks like pearl which indicates Iyam prominate in 65% of cases.

In the balance 35% it spreads slowly and uniformly.

Naadi

In all the patient kapha thondha naadi is prominent

(ie) Kapha Pitham 30%

Kapha vatham 50%

Vatha Kapham 20%

“nr¤Jk ÓjkhŒ Jil¤J”

Than Iyam is prominent in later stage of life.

Signs and Symptoms:

The main symptoms of madumegam polyuria, Polyphagia, Polydipsia, Glycosuria and emaciation are present in all cases.

It has marked improvement in all the above symptoms clinically and their condition was good.

Urine sugar fasting and urine sugar post prandial has become normal in 80% of the cases.

Blood sugar post prandial level has become normal for 70% of the cases.

The post prandial blood sugar below 200 mgs show good response.

The post prandial blood sugar in the range of 200-249 my shows moderate response.

The post prandial blood sugar above 250 mg shows mild response.

The biochemical studies of Sarabenthra Madhumega Choornam shows it contains starch and tannic acid which prevent over correction leading to

hypoglycemia. The drug shows no side effects and contra indication. The drug was also subjected to Pharmacological and toxicological tests in rat models. The results revealed the combination of Sarabenthra Madhumega Choornam and Madhumega Kudineer has very effective results. There were no signs of toxicity as could be judged by the absence of undesirable clinical manifestations.

The prevalence of the disease is mainly attributed to the fast changing lifestyle full of stress. The concept of "Unavathi Seyal" as the primary cause of disease as mentioned by Siddhars has become very relevant in the current context.

SUMMARY

Madhumegam is one of the 20 varieties of Megarogam, as classified and explained by Siddhar Yogi Munivar. Moreover, Madhumegam which comes under Azhalneer is very specific and correlates with Maturity onset diabetes mellitus be i.e. non-insulin dependent, which is a chronic metabolic disorder. Currently Madhumegam is considered as one of the worst life style disorders faced by civilized world.

The disease Madhumegam has been thoroughly studied by selecting 20 in patients for a period of 6 weeks in Post Graduate branch I Maruthuvam department, Government Siddha Medical College and Arignar Anna Hospital, Chennai – 600106.

Patients were examined with Siddha and modern concepts at the post graduate department, Branch I Pothu Maruthuvam.

The medicine administered were

1) Madhumega Choornam 1 gm t.d.s. with hot water after food.
2) Madhumega Kudineer – 30 ml b.d. before food.

CONCLUSION

- The selected drugs used in the study are easily available.
- The preparation of the trial drug is simple.
- The drug is palatable and hence the oral route of administration is easy.
- The ingredients of the drug are available in plenty in the rural areas, poor people living there may have the maximum benefit.
- The pharmacological study revealed that the Choornam and Kudineer in the combined from yielded good results in rat models and there was no toxicity in the drug.
- It may be noted that the drug is a safe and effective one for Madhumegam.
- No hypoglycemic complications were observed during the study.
- Hence it could be administered even for longer period if required

So it is concluded the combined theraphy with Sarabenthra Madhumega Choornam and Madhumega Kudineer can be very good in the view of efficacy and safety in Madhumegam.

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Murungaipattai

Maruthampattai

Sarakondaraipattai

Karuvelmpisin

Aathipattai

Avarampattai

Salamisiri

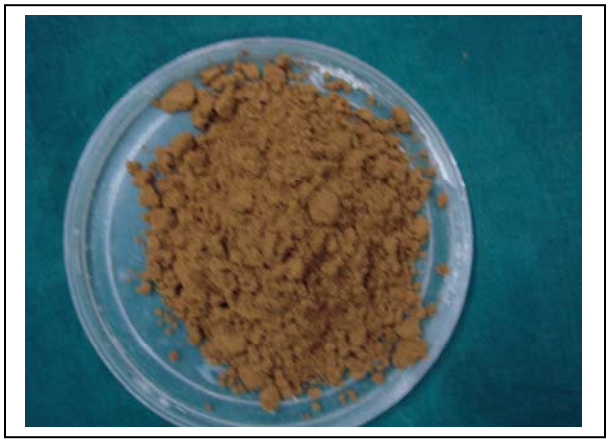
Madhumega Kudineer Coarsely powder

Manjal

Maruthani Vithai

Karunesembaipattai

Kothimai Sathu



Sarabenthra Madhumega Choornam

hS.No.	In Patients No.	Signs & Symptoms	Urine			Motion		Haematological Report								
				Alb	Dep	Ova	Cyst	TC Cu.mm	DC			ESR [Hour]		Hb gms	Urea mg	Cholesterol mg
									P	L	E	½	1			
1	1840 / 9261	Polyuria, Poly Phagia, Poly dipsia, Pain all over the body, constipation	B/T	Nil	Nil	Nil	Nil	9400	59%	35%	6%	24	40	10	29	182
			A/T	Nil	Nil	Nil	Nil	9800	61%	36%	4%	10	20	11	27	180
2	1795 / 7185	Pain all over the body, polyuria, Poly Phagia, Dryness of the mouths throat, Dry Skin	B/T	Nil	Few Pus cells	Nil	Nil	9800	57%	38%	5%	20	40	10	22	180
			A/T	Nil	Nil	Nil	Nil	9700	59%	38%	3%	12	24	10	22	175
3	1792 / 7091	Poly Uria, Poly Phagia, Poly Dipsia, Constipation, Peripheral Neuritis.	B/T	Nil	Nil	Nil	Nil	9800	55%	41%	4%	24	40	9	26	179
			A/T	Nil	Nil	Nil	Nil	9800	55%	45%	2%	18	36	11	24	170
4	1949 / 4952	Polyuria, Polyphagia, Pruritis Valvae, pain all over the body, Exaciation	B/T	Nil	Nil	Nil	Nil	9000	53%	35%	5%	10	12	8	27	208
			A/T	Nil	Nil	Nil	Nil	9200	51%	36%	3	10	12	9	25	200
5	1993 / 6854	Poly Uria, Poly Phagia, Itching all over the body, Dry Skin, Diabetic foot ulcer	B/T	Nil	Nil	Nil	Nil	9700	60%	34%	6%	25	54	7	21	177
			A/T	Nil	Nil	Nil	Nil	9600	60%	32%	4%	18	36	9	21	170
6	2436 / 2610	Poly uria, Poly Phagia, Pain all over the body, Dryness of the mouth and throat, constitpation	B/T	Nil	Nil	Nil	Nil	10000	57%	38%	5%	12	20	10	21	170
			A/T	Nil	Nil	Nil	Nil	10200	59%	40%	3%	10	20	11	21	168
7	2070 / 1413	Poly Uria, Poly Phagia, Dry skin, Emaciation, Peripheral Neurits	B/T	Nil	Few Pus cells	Nil	Nil	9800	59%	35%	4%	15	34	10	27	185
			A/T	Nil	Nil	Nil	Nil	9800	61%	35%	3%	10	20	10	25	180
8	2175 / 8747	Poly Phagia, Polydipsia, Poly uria, pain all over the body, constipation	B/T	Nil	Nil	Nil	Nil	10200	66%	41%	6%	24	40	9	23	178
			A/T	Nil	Nil	Nil	Nil	10400	67%	42%	4%	10	20	8	23	170
10	2166 / 8110	Poly uria, Poly Phagia, Pruritis vulvae, Dryness of the mouth an throat	B/T	Nil	Nil	Nil	Nil	9400	58%	36%	9%	12	20	11	25	159
			A/T	Nil	Nil	Nil	Nil	9800	60%	38%	3%	12	20	12	25	150

S. No.	In Patients No.	Signs & Symptoms	Urine			Motion		Haematological Report								
				Alb	Dep	Ova	Cyst	TC Cu.mm	DC			ESR [Hour]		Hb gms	Urea mg	Cholesterol mg
									P	L	E	½	1			
11	2412 / 9446	Poly uria, Polyphagia, Pain all over the body, Emaciation, constipation	B/T	Nil	Few Pus cells	Nil	Nil	10400	62%	33%	5%	20	40	9.5	27	158
			A/T	Nil	Nil	Nil	Nil	10800	64%	31%	3%	10	20	10	25	155
12	2446 / 874	Poly Phagia, Poly uria, Dryness of the Mouth and throat, constipation	B/T	Nil	Nil	Nil	Nil	9800	59%	35%	4%	10	12	11	27	172
			A/T	Nil	Nil	Nil	Nil	9700	57%	36%	1%	10	12	11	25	170
13	2438 / 213	Poly Uria, Poly Phagia, Itching all over the body, Dry skin, Emaciation	B/T	Nil	Nil	Nil	Nil	9700	58%	35%	7%	20	45	9	24	162
			A/T	Nil	Nil	Nil	Nil	9800	59%	33%	4%	12	24	10	24	162
14	2416 / 9768	Poly Uria, Poly Phagia, pain all over the body, Dryness of the Mouth	B/T	Nil	Nil	Nil	Nil	8000	53%	42%	6%	44	80	8.5	24	165
			A/T	Nil	Nil	Nil	Nil	8100	55%	42%	4%	20	40	10	22	160
15	2209 /24	Poly Uria, Poly Phagia, Pruritis Vulvae, Itching all over the body, constipation	B/T	Nil	Nil	Nil	Nil	10600	64%	31%	5%	42	80	10	24	172
			A/T	Nil	Few Pus cells	Nil	Nil	10800	66%	34%	1%	20	40	11	21	170
16	2571 / 7785	Poly Uria, Poly Phagia, Pain all over the body, peripheral neuritis, Diabetic footulcer	B/T	Nil	Nil	Nil	Nil	10200	60%	34%	6%	30	60	11.08	20	212
			A/T	Nil	Nil	Nil	Nil	10400	61%	35%	3%	18	36	12	20	210
17	2726 / 6440	Poly Uria, Poly Phagia, pain all over the body, Emaciation	B/T	Nil	Few Epi. Cells	Nil	Nil	10000	67%	20%	5%	25	53	10.5	23	210
			A/T	Nil	Nil	Nil	Nil	10000	67%	24%	3%	10	36	11	21	200
18	2577 / 4281	Poly Uria, Poly Dipsia, Poly Phagia, Itching all over the body.	B/T	Nil	Nil	Nil	Nil	9400	57%	38%	5%	12	20	9.5	26	191
			A/T	Nil	Nil	Nil	Nil	9200	55%	34%	3%	12	20	11	24	190

S. No.	In Patients No.	Signs & Symptoms	Urine			Motion		Haematological Report								
				Alb	Dep	Ova	Cyst	TC Cu.mm	DC			ESR [Hour]		Hb gms	Urea mg	Cholesterol mg
									P	L	E	½	1			
19	2657 / 2743	Poly Uria, Poly Phagia, Pain all over the body, constipation	B/T	Nil	Few Pus cells	Nil	Nil	9000	56%	34%	5%	12	20	10	29	159
			A/T	Nil	Nil	Nil	Nil	9200	59%	35%	1%	12	20	11	27	155
20	2760 / 9140	Poly Uria, Poly Phagia, Dryness of the mouth and throat, constipation	B/T	Nil	Nil	Nil	Nil	9400	59%	34%	7%	42	80	10	25	182
			A/T	Nil	Nil	Nil	Nil	9800	61%	36%	4%	20	40	12	25	189
21	2840 / 1876	Poly Uria, Poly Phagia, peripheral Neuritis, Pain all over the body, Dry Skin	B/T	Nil	Nil	Nil	Nil	9700	55%	34%	6%	12	25	11.5	22	205
			A/T	Nil	Nil	Nil	Nil	9800	59%	38%	3%	10	18	12	22	200

Statement of out – Patient cases for the disesases 'MADHUMEGAM"

S. No	Name of the Patients	Age	Sex	OP. No.	In Come	Diet	F/H	Nature of Job	BB	Date		Urine Sugar		BloodSugar mg%	
										From	To	BT	AT	BT	AT
1	Mani	49	M	8860	400	NV	Father	AL	OW	17.10.07	30.11.07	++	Nil	256	130
2	Vishvanathan	38	M	4840	400	NV	G.Father	AL	OW	06.12.07	20.01.08	++	Nil	243	120
3	Lakshmi	60	F	8004	400	NV	Paternal	HW	OW	15.12.07	20.01.08	+	Nil	149	100
4	Papathi	56	F	7986	400	NV	Father	HW	OB	15.12.07	30.01.08	++	+	175	170
5	Valli	50	F	8218	500	NV	Mother	HW	OB	25.12.07	05.02.08	+++	Nil	279	140
6	Perumal	65	M	8305	500	VEG	G.Father	AL	N	25.12.07	27.01.08	++	Nil	197	130
7	Bagubali	67	M	1646	500	NV	Paternal	AL	OW	26.12.07	07.02.08	++	Nil	226	160
8	Sumathi	41	F	155	500	NV	Father	FS	OB	27.01.06	10.03.08	+	Nil	182	140
9	Brushothaman	62	M	810	500	NV	Mother	AL	OB	24.01.08	30.02.08	++	Nil	243	160
10	Nadarajan	42	M	3249	500	NV	Paternal	AL	N	27.02.08	10.04.08	++	Nil	261	160
11	Sonal	78	M	2569	500	NV	G.Father	AL	N	29.01.08	10.03.08	+	Nil	170	130
12	Palani	37	M	5258	500	NV	Paternal	AL	OW	05.02.08	10.02.08	++	Nil	264	160
13	Aathimoolam	65	M	3350	400	VEG	Father	WM	OW	26.02.08	10.04.08	++	Nil	217	150
14	Sulochana	60	F	2940	400	NV	Mother	HW	OB	25.02.08	05.04.08	++	++	200	180
15	Chinaaponnu	38	F	5934	500	NV	Father	HW	OB	08.01.08	20.02.08	++	Nil	242	160
16	Muthu	50	M	749	500	NV	Mother	AL	OW	29.01.08	10.03.08	++	Nil	238	150
17	Govindhasami	65	M	7119	500	VEG	G.Father	C	OW	11.01.08	13.02.08	++	Nil	210	130
18	Padmanaban	53	M	3976	500	NV	Father	AL	N	02.02.08	15.03.08	+	Nil	148	110
19	Saravanakumar	32	M	3737	500	NV	Mother	AL	OB	01.02.08	15.03.08	++	Nil	268	140
20	Kasiammal	48	F	5936	500	NV	G.Father	FS	OW	04.03.08	15.04.05	++	Nil	183	120

Statement of out – Patient cases for the disesases 'MADHUMEGAM"

IP. No.	Name	Ward	Age	Sex	Red	DOA	DOD	INC	DIET	F/H	Nature of Job	BB	Urine Sugar Fasting		Urine Sugar – PP		Blood Sugar Admission		Blood Sugar Discharge	
													BT	AT	BT	AT	BT	AT	BT	AT
1840 / 9261	Vadivelu	IXMMSS	65	M	H	21.11.07	05.01.08	500	NV	Father	AL	OW	++	Nil	++	Nil	148	200	110	160
1795/ 7185	Saroja	IVOGHS	48	F	H	14.11.07	26.12.07	500	NV	Maternal	HW	OW	++	Nil	++	Nil	140	268	110	140
1792 / 7011	Jeeva	IVOGHS	39	F	H	14.11.07	22.12.07	400	NV	Father	HW	OB	++	Nil	++	Nil	130	186	100	140
1949 / 4952	Sadaiyani	10SPMCS	65	M	H	06.12.07	21.01.08	400	NV	Paternal	AL	OB	+	Nil	+	Nil	140	270	90	150
1993 / 6854	Arukkani	XIEMCS	40	F	H	12.12.07	02.01.08	400	NV	Mother	HW	N	++	+	++	+	160	219	160	200
2436 / 2610	Cinnaponnu	X FMCS	38	F	H	17.12.07	10.01.08	500	NV	Maternal	HW	OW	+	Nil	+	Nil	120	299	80	145
2070 / 1413	Govindammal	IV OGHS	35	F	H	26.12.07	11.01.08	500	VEG	Father	HW	OW	+	Nil	+	Nil	148	211	98	133
2175 / 8747	Menakshi	XI FMCS	60	F	H	18.01.08	10.02.08	500	NV	G.Father	HW	OB	++	Nil	+++	Nil	159	268	88	158
2166 / 8110	Suresh	X MSPMCS	32	M	H	16.01.08	02.02.08	400	VEG	Paternal	FS	N	++	+	++	+	132	170	120	140
2412 / 9446	Maniammal	IV OGHS	52	F	H	16.02.08	05.03.08	400	NV	Mother	HW	OW	++	Nil	++	Nil	136	162	81	120
2446 / 874	Anbalagan	10SPMCS	44	M	H	20.02.08	01.04.08	400	NV	Father	AL	OW	++	Nil	++	Nil	119	280	79	141
2438 / 2132	Souriammal	XI FMCS	58	F	H	18.02.08	15.03.08	500	NV	NRH	HW	OW	++	Nil	+	Nil	120	170	110	140

IP. No.	Name	Ward	Age	Sex	Red	DOA	DOD	INC	DIET	F/H	Nature of Job	BB	Urine Sugar Fasting		Urine Sugar – PP		Blood Sugar Admission		Blood Sugar Discharge	
													BT	AT	BT	AT	BT	AT	BT	AT
2416 / 9768	Ponnusami	X SPMCS	52	M	H	18.08.06	15.04.08	500	NV	Mother	WM	OB	++	Nil	++	Nil	152	213	96	158
2209 / 2413	Jothi	IV OGHS	68	F	H	22.03.08	20.04.09	500	NV	NRH	FS	OW	++	Nil	++	Nil	138	210	80	150
2571 / 7785	Krishna Moorthi	X SPMCS	35	M	H	10.03.08	16.04.08	500	NV	Father	AL	OW	++	Nil	++	Nil	135	162	90	100
2726 / 6440	Subedha Begam	XI FMCS	85	F	H	01.03.08	05.04.08	400	NV	Maternal	HW	OW	++	++	++	++	140	247	130	240
2577 / 4281	Alamelu	XI FMCS	57	F	H	29.03.08	25.04.08	500	NV	Mother	HW	OB	++	Nil	++	Nil	119	210	79	120
2657 / 2743	Govindammal	SFMC	65	F	H	24.03.08	20.04.08	500	VEG	G.Father	HW	OB	++	Nil	++	Nil	120	168	96	112
2760 / 9140	Pondurangen	SPMHS	71	M	H	08.04.08	05.05.08	400	NV	Father	WM	OW	++	+	++	+	151	210	135	190
2840 / 1876	Arumugam	SPMHS	60	M	H	16.04.08	10.05.08	500	NV	NRH	C	N	+	Nil	+	Nil	159	180	81	106

Red - Religon H – Hindu M – Muslim C – Christian BB – Body Build

OW - Over Weight OB – Obese N – Normal HW – House Wife

FS - Flower Seller Al – Agricultural Labour WM – Watchman

NV - Non Vegetarian C – Carpenter NRH – No relevant history